THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI –TAMILNADU

DISSERTATION ON

" STUDY ON SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION "

SUBMITTED FOR

MD DEGREE EXAMINATION

BRANCH 1 (GENERAL MEDICINE)

EXAMINATION IN

APRIL 2016

THANJAVUR MEDICAL COLLEGE

THANJAVUR

CERTIFICATE

This is to certify that this dissertation entitled **" A STUDY ON SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION"** is a bonafide record work done by **Dr.ALEX BABY** in the Department of General Medicine,Thanjavur Medical College,Thanjavur during his Post Graduate Course from 2013 – 2016.This is submitted as partial fulfillment for the requirement of M.D. Degree Examination, General Medicine (Branch 1) to be held in April 2016.

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DECLARATION

I, Dr. ALEX BABY, solemnly declare that dissertation titled " A STUDY ON SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION " is a bonafide work done by me at Thanjavur Medical College Hospital during July 2014 to May 2015 under the guidance and supervision of Prof.Dr.K.NAMASIVAYAM M.D.. The dissertation is submitted to THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY, CHENNAI, TAMILNADU as partial fulfillment for the requirement of M.D. Degree Examination – Branch 1 (General Medicine) to be held in April 2016.

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CONTENTS

LIST OF ABBREVIATIONS

IHD	ISCHEMIC HEART DISEASE
TG	TRIGLYCERIDES
LPL	LIPOPROTEIN LIPASE
MAT	MULTIFOCAL ATRIAL TACHYCARDIA
VT	VENTRICULAR TACHYCARDIA
VF	VENTRICULAR FIBRILLATION
STEMI	ST ELEVATION MI
NSTEMI	NON ST ELEVATION MI
HDL	HIGH DENSITY LIPOPROTEIN
LDL	LOW DENSITY LIPOPROTEIN
Mg	MAGNESIUM
MGSO4	MAGNESIUM SULPHATE
ATP	ADENOSINE TRI PHOSPHATE
VA	VENTRICULAR ARRYTHMIAS

NO	NITRIC OXIDE
MI	MYOCARDIAL INFARCTION
AV	ATRIO VENTRICULAR
SA	SINO ATRIAL
VLDL	VERY LOW DENSITY LIPOPROTEIN
ATN	ACUTE TUBULAR NECROSIS
CAD	CORONARY ARTERY DISEASE
CCF	CONGESTIVE CARDIAC FAILURE
DTR	DEEP TENDON REFLEX
GFR	GLOMERULAR FILTERATION RATE
PEM	PROTEIN ENERGY MALNUTRITION.

ABSTRACT AND KEYWORDS

BACKGROUND OF STUDY: Magnesium has been implicated in the pathogenesis of myocardial infarction and its complication like arrhythmia. Magnesium improves myocardial metabolism , inhibits calcium accumulation and myocardial cell death. It improves vascular tone, peripheral vascular resistance, after load and cardiac output, reduces cardiac arrhythmias and improves lipid metabolism. Magnesium also reduces vulnerability to oxygen derived free radicals, improves endothelial function and inhibits platelet aggregation and adhesion.

OBJECTIVE: To know the relationship between the serum magnesium levels and arrhythmias in patients with acute myocardial infarction.

METHOD: By using simple random method, 60 cases of acute myocardial infarction admitted in THANJAVUR MEDICAL COLLEGE HOSPITAL, THANJAVUR over a period of 11 months, i.e., between July 2014 to may 2015.

RESULTS: There is significant difference in Magnesium levels in patients with and without arrhythmias.

CONCLUSION: In acute myocardial infarction, patients with low magnesium levels are more prone to get arrhythmias. So magnesium treatment can be considered in patients of acute myocardial infarction with low magnesium levels.

KEYWORDS: Magnesium; Myocardial Infarction ; Arrythmias.

INTRODUCTION

Pasteur (1860) showed that yeast will grow only when the culture medium contains inorganic compounds. In the human body there is a tendency to maintain the proper fluid balance, not only as a whole but between the three compartments of intracellular, interstitial and intravascular spaces. This is maintained by an intricate play of hemodynamic, electrolyte and other forces.

The field of mineral metabolism is at present in a phase of rapid expansion. It has become apparent that not only proteins, fats and carbohydrates, but also minerals are essential to life. Now the significance of traces not only of vitamins and other active organic substances, but also of minerals is under intensive investigation. Magnesium has been implicated in the pathogenesis of Acute Myocardial Iinfarction and its complications like arrhythmias. It plays a significant role in other cardiovascular diseases as well. Magnesium ions are considered essential for the maintenance of the functional integrity of the myocardium.

Myocardial magnesium concentration in patients with sudden death due to ischemic heart disease was found to be very low . It has been pointed out that magnesium has a vital role in ventricular fibrillation, which causes sudden death in IHD. The coronary vasospasm resulting from magnesium deficiency has been suggested as another important factor in the sudden death in IHD. Magnesium deficiency was also postulated to have role in the genesis of atheromatous plaques in

that it leads to hyperlipidemia. Also myocardial infarction is one of the common causes of death at present where prognosis depends on multiple factor of which many still remain unexplained. This study is designed to know the relationship between serum magnesium levels and arrhythmias in patients with acute myocardial infarction.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

To know the relation between level of serum magnesium and arrhythmias in patients with acute myocardial infarction who are presenting within 12 hours of onset of symptoms**.**

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORY REVIEW

Inorganic constituents forms only a small part of human body, yet they are very essential for sustaining life. It was Liebig (1803-1873) who recognized the importance of minerals as vital parts of plants and animals. The name 'Magnesium' was given by Sir Humphrey Davy . It was obtained from the word 'Magnesia' which was an ancient Gracian town. Magnesium(mg) was discovered by Joseph Black in 1755.He distinguished Magnesium oxide (MgO) from Calcium oxide (CaO). Meerschaum (Magnesium Silicate) was found out by Thomas Henry in 1789. French scientist Antoine – Alexandre – Brutus - Bussy reacted magnesium chloride with potassium and obtained a sizeable amount of magnesium and carried out many studies on Magnesium.

Greenberg described myocardial degeneration with polyplastic infiltration and fibroblast proliferation in rats, who were fed on a low magnesium diet. Magnesium is an essential component of enzymes involved in oxidative phosphorylation.The abnormalities observed in rats were found to be sequelae to interference of function of these magnesium dependant enzymes.[1]

Until middle of Twentieth centuary, isolation and estimation of magnesium was difficult and lacked uniformity in procedures. So the studies on magnesium and its effects on human body were neglected for long time. But reasonable amount of work on

significance of magnesium in non primates were available. The availability of more accurate uniform methods for estimation of serum magnesium in laboratory gave momentum to work on magnesium metabolism in man.

Only 1% of total body magnesium is in the extra-cellular fluid and of this about 25% is in the plasma, rest is in the red cells. Around 50% of serum magnesium is free, 32% is protein bound and rest 13% is accounted for magnesium phosphate, citrate and other unidentified complexes. The vascular space constitutes a minor content of magnesium concentration in the body, so the estimation of plasma concentration of ,Mg doesnot always impact the actual concentration of Mg in patients , but intracellular estimation of Mg levels are under research and not popularly available. (Vermon et al, 1978)[3].

MAGNESIUM METABOLISM

Manesium is the second most abundant cation in the Intra cellular fluid , after Potassium[5] . It ranks fourth among other cations regarding abundance in the human body. Magnesium is not uniformly distributed among tissues of human body. Magnesium is distributed in Human body according to the metabolic activity of tissues. Maximum concentration of magnesium is found in Heart , Kidneys and Brain. Bone contains more than 50% of total body Magnesium content and this forms a exchangeable source that maintains normal serum magnesium.Only one third of total body bone magnesium is in exchangeable form[6].

Normal human adult body contains 20 – 24 gram of magnesium or approximately 2000 milliequivalents(meq) of magnesium. Of this only 1% is found in the extracellular fluid.The normal serum magnesium level is found to be 1.8 – 2.9 mg/dl[7]. Only 25% of magnesium in extracellular fluid is found in plasma. Of the total plasma magnesium about 70% is ultrafilterable. Free form of magnesium constitutes about 50%.Bound form of plasma magnesium is bound to plasma proteins mainly Albumin. Cellular magnesium levels in tissues varies with metabolic activity of tissues.Higher the metabolic activity higher the levels of magnesium. The normal cellular magnesium level is between 1 – 3 mmol/L[8]. Intracellular magnesium levels maynot always correlate with serum magnesium levels, because only 25% of total extracellular magnesium is found in serum[9]. But cellular assays of Magnesium are not reliable and not widely

available. So serum magnesium estimation remains as the best method to evaluate magnesium deficiency or magnesium excess.

RENAL MAGNESIUM HANDLING

The principal organ involved in the regulation of Magnesium Homeostasis is kidney. Every day approximately 8 meq is excreted in urine. During periods of Mg deficiency renal conservation of Mg occurs and it excretes very low amount of Mg. But when in states of Mg abundance, kidney excretes more amount of Mg to maintain Mg balance in the human body [10]. In humans handling of magnesium by kidneys is a filteration – reabsorption process .Micropuncture studies have shown that proximal convoluted tubule and thick ascending loop of henle are the major sites of magnesium reabsorption. 20 to 30 % of filtered magnesium is reabsorbed passively in the proximal tubules where magnesium reabsorption follows change in the salt and water reabsorption and is associated with the rate of fluid flow. Of the total magnesium filtered about 65% is reclaimed in the thick ascending loop of henle by an active transport process.

Parathyroid hormone regulates, in part, both calcium and magnesium metabolism and excretion. Parathormone reduces magnesium excretion where as aldosterone increase renal excretion of magnesium. Parathormone doesnot have important physiological role in the regulation of serum magnesium level as patients with both hypoparathyroidism and hyperparathyroidism have normal serum magnesium concentration.

MYOCARDIAL MAGNESIUM

Most of the magnesium in human body is found in bone.The adult human body contains between 20 and 30 gms of magnesium.Cardiac muscles also has significant concentration of magnesium.The higher concentration of magnesium is found in ventricles than in atria.There is no significant change in the amount of magnesium between right and left ventricles or the interventricular septum[1]. Magnesium was found to be involved in the ATP hydrolysis of myofibrils, super-precipitation and sineresis of actinomycin gels, and binding and release of calcium ions by sarcotubule reactions, which are essential to the contraction of heart muscle.Magnesium activates adenyl cyclase, stimulates oxidative phosphorylation in heart mitochondria and affect sodium potassium ATPase of heart membranes.Though myocardium is less sensitive than nervous tissue to magnesium, it may have influenze in muscle tone and conducting system.[5]

MAGNESIUM ABSORPTION FROM INTESTINES.

The minimum recommended daily requirement of magnesium is 300- 500 mg . The intestinal absorption is inversely proportional to the amount ingested[11]. The usual daily magnesium intake is between 150-350 mg/day. About 50 % of ingested magnesium is reabsorbed[12]. The major sources of magnesium are green leafy vegetables ,meat ,cereals and nuts.

Magnesium is absorbed maximally from ileum and jejnum, though it is absorbed along entire intestinal tract. Magnesium is absorbed most efficiently in the chloride form in the alkaline environment of small intestine[13]. The existence of an unsuitable passive transport system for magnesium absorption may account for the higher fractional absorption at low dietary magnesium intake.

A principle factor of hormone controlling intestinal magnesium transport has now been described. 25 hydroxy vitamin D and 1,25 dihydroxy vitamin D have been found to enhance magnesium absorption by the intestine[14]. Bio availability of magnesium may also be a factor in magnesium intestinal absorption. Excessive amounts of substances such as free fatty acids, phytates, oxalates, phosphate and fiber may bind magnesium and impair absorption[5].

INTRACELLULAR MAGNESIUM

Magnesium is bound to proteins and negatively charged molecules inside the cell. Significant amounts of magnesium are found in the nucleus ,mitochondria and endoplasmic reticulum as well as cytoplasm. 80 % of magnesium in the cytoplasm is complexed with adenosine triphosphate(ATP)[11]. The concentration of free ionized magnesium is about 0.1 mmol/l to 1 mmol/l. It constitutes about 0.55 to 5 % of total cellular magnesium. The magnesium concentration in the cell cytoplasm appears to be maintained relatively constant.

PHYSIOLOGICAL ROLE OF MAGNESIUM

It is essential for substrate formation and has direct role in the activation of enzymes such as phosphofructokinase, creatinine kinase, adenylate cyclase and sodium-potassium ATPase[5]. Magnesium plays a role in numerous enzymatic processes in the body[5]. Magnesium has important roles in biological processes such as glycolysis,oxidative phosphprylation, nucleotide metabolism, protein biosynthesis signifies the importance of magnesium in cellular metabolism.

Observation in humans have shown that magnesium deficiency impairs the cells ability to maintain potassium gradient resulting in intracellular potassium depletion. Magnesium activates the sodium potassium ATPase, which inturn maintain high intracellular and low extracellular potassium levels against large concentration gradients. The compromised cell membrane cation pump causes loss of cellular potassium and accumulation of intracellular sodium. This effect is similar to that which occurs during digitalis therapy and may explain why magnesium deficiency enhances digitalis toxicity.

MAGNESIUM : EFFECT ON CARDIAC RHYTHM

Magnesium has modest electrophysiologic effects. Intracellular hypokalemia, hyponatremia and increase of cell excitability may be associated with magnesium deficiency. Magnesium has effect on real and adjused SA node conduction time, increases AV conduction time, has effect on relative refractory periods, during ventricular pacing QRS duration is extended lengths of cycle more than 250

milliseconds[46]. Zwillinger[31] was the first person to discover the effect of Mg on arrhythmias and was later used widely in the management of arrhythmias and usefully employed in patients with difficult VTs, VAs induced by digitalis and in those with episodes of TdP.

Magnesium was found to be useful in a variety of arrhythmias including both tachy and brady arrhythmias and was employed widely in clinical practice for the same purpose. Recently many studies have proposed Mg as the most useful drug in various life threatening arrhythmias[47]. .

Magnesium is the natural calcium antagonist.Magnesium reduces calcium influx to the cells during ischemia and prevents damage associated with ischemia to the myocardium. Magnesium also affects systemic and pulmonary vascular impedence and causes decrease in many hemodynamic parameters.

Impact of Magnesium on Lipid Metabolism

Magnesium has an unproven role in the metabolism of lipids.Magnesium therapy showed improvement in the cholesterol metabolism such as improvement in the ratio of HDL-C and LDL-C plus VLDL- C.The levels of platelet magnesium levels intracellulary has a great impact on the role of platelets in causing thrombosis or atherogenesis. In a study non rabbits these organisms were fed on a special diet with high amount of cholesterol and different amounts of magnesium and was found that there was a dose dependant decrease in aortic lesions and cholesterol content of lesions[48]. A study on rats fed with

similar diet but showed adverse effects . In another study on rats the amount of cholesterol content ,LDL and TGs associated with a reduction in the levels of good cholesterol.[49].

Rassmussen et al [51] found that when diets containing Magnesium 15 mmol/day showed a significant reduction in the plasma levels of TGLs and VLDL-C and a significant reduction in plasma levels of good cholesterol.

Mg was found to be very important part of many of the biomolecules involved in cholesterol metabolism

Another study showed that amont of Mg in thrombocytes significantly related inversely to the levels of total cholesterol. As a result, the low levels of platelet cellular magnesium levels can be considered as apossible marker of alterations of the membrane of platelets and its involvement in thrombosis and atherosclerosis[52].

Anticoagulant/ Antiplatelet Properties of Magnesium

Many studies have shown that addition of Mg to the fresh notclotted plasma had caused an increase in the clotting time. During the world wars , in germany it was found that when magnesium was used as a muscle relaxant in wounded patients it was observed that after death during postmortem their blood didnot clot [53].It was later proved that magnesium inhibits human blood clotting[54].

Many studies have proven the importance of magnesium in vascular complications like myocardial infarctions and cerebrovascular events. Mg prevents the growth of thrombus and thus facilitates the opening of the involved coronaries and improves the mortality benefit of patients in various studies[55,56]. Magnesium was found to be most important cation involved in thrombus formation after calcium.

Very high concentration of magnesium has effects on regulating various factors involved in the coagulation cascade. Mg has important roles in the pathogenesis of atherothrombosis in MI and has a proven role in the prevention of arrhythmias associated with myocardial infarction. Mg has effects like inactivation of various factors involved in the aggregation and activation of platelets and thus prevents thrombus formation[57,58]. Even administration of magnesium to control subjects who donot have the low serum magnesium level was found to be beneficial to this regard. So the crucial role of magnesium, in the pathogenesis and prevention of coronary thrombosis and the associated arrhythmias is undisputable. This also underlines the correction of low magnesium levels in patients with vascular complications and the improvement of prognosis associated with these conditions.

ROLE OF MAGNESIUM ON ENDOTHELIAL FUNCTION:

Many studies have shown a crucial role for magnesium on endothelial function. Decreased serum Mg has been found to impair NO synthesis. The decreased production of Endothelial nitric oxide leads to decreased vasodilatation and this predisposes to vascular thrombosis and endocardial ischemia. The impairment of endothelial function associated with magnesium deficiency is proposed to be be the reason for increased predisposition for myocardial ischemia in patients with hypomagnesemia.So the correction of low serum magnesium levels in these patients is postulated to improve the prognosis of these patients and decrease the mortality.

CORONARY CIRCULATION

ANATOMY OF CORONARY ARTERIES

Blood supply of heart is derived from right and left coronary arteries which arise from anterior aortic and left posterior aortic sinus respectively.

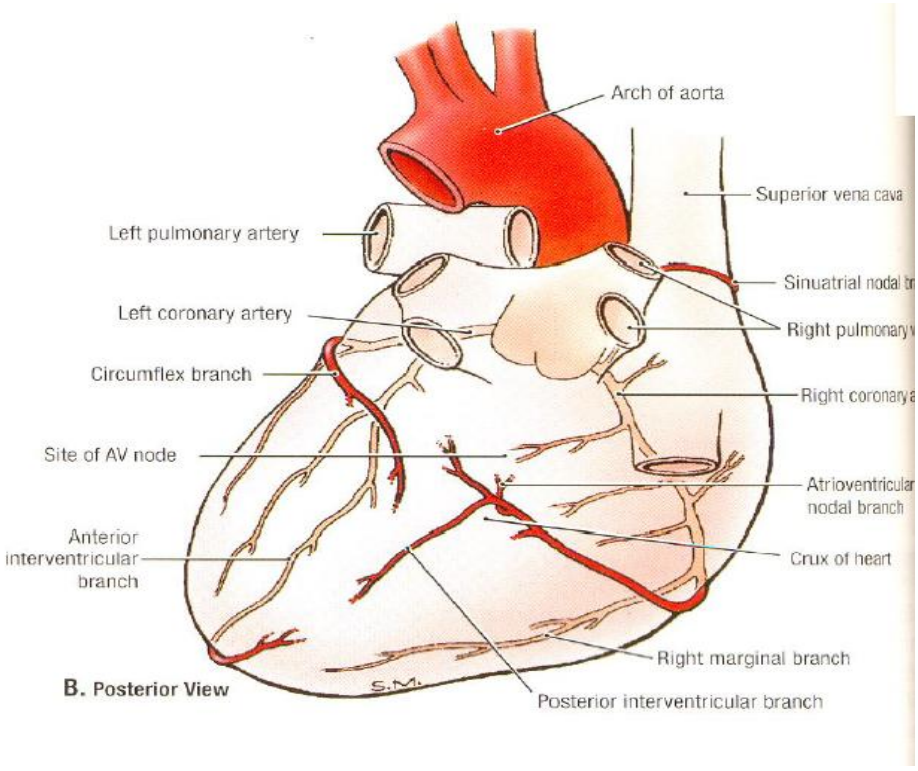
Right coronary artery after arising from the anterior sinus passes between the right auricular appendage and infundibulum of right ventricle. Passing now vertically downwards in the atrioventricular groove the artery turns backwards at the inferior border of the heart and runs posteriorly. It gives off branches to both atria and ventricles as it passes vertically downwards. At the inferior border, the marginal branch passes to the left along the right ventricle. On the diaphragmatic surface, the inferior interventricular branch is given off. This large artery passes along the interventricular groove to the apex of the heart. The terminal part anastomoses with the terminal arterioles of the coronary artery at the lower part of the left atrium.

The left coronary artery immediately after its origin divides into anterior descending artery and left circumflex artery. The anterior descending artery runs in the interventricular groove to anastomose at the apex with the terminal branches of the inferior interventricular artery. The left circumflex gives off branches to the posterior wall of the left ventricle and runs on to anastomose with the termination of the right coronary artery, below the coronary sinus.

CORONARY CIRCULATION

Figure A

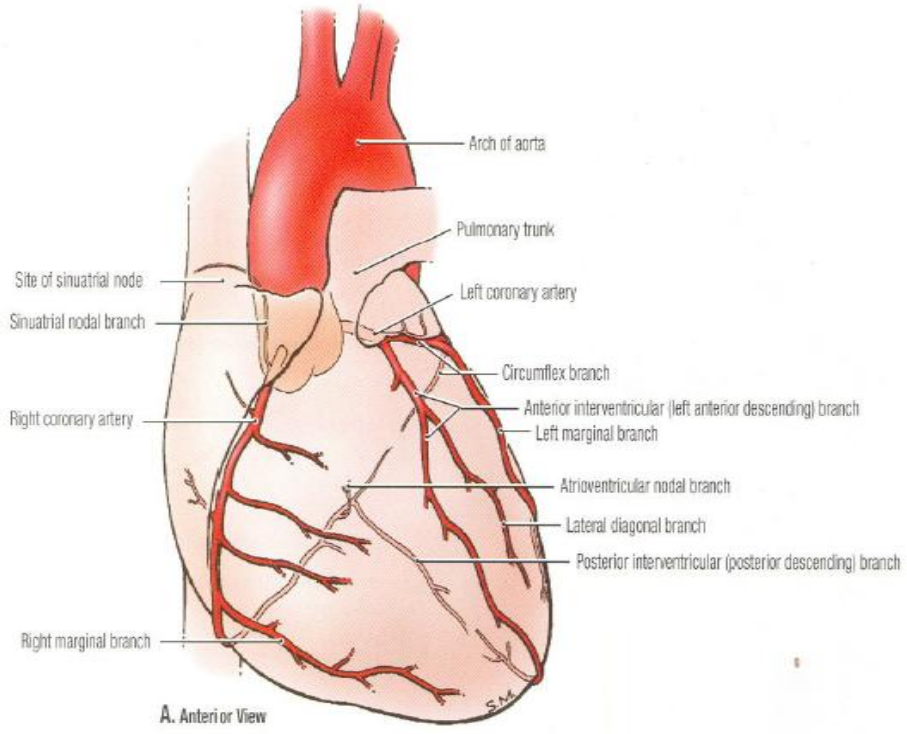
The blood supply to heart is through coronary arteries. Right coronary artery (RCA) is originated from the great vessel Aorta, from its anterior sinus. The first part of RCA moves in between the main pulmonary artery and the right atrium. The second part of the RCA runs over the sternal part of the heart moving between the RV and RA. The third part is formed at the lower part of heart moves in the AV groove.



CORONARY CIRCULATION

Figure B

Left coronary artery (LCA) is from the sinus of the great vessel aorta which is the posterior sinus .It moves between the Main pulmonary artery and the left atrial chamber of heart. Then the LCA moves into the Sternal surface of heart and forms two channels Circumflex artery and another artery anterior interventricular artery. The circumflex artery moves in the part of the groove separating atrium and ventricle in its anterior part.



ANASTOMOSES OF CORONARY ARTERIES

Anastomoses exists between the terminations of right and left coronary arteries in the atrrioventricular groove and these surface anastomoes are insignificant. There are intercoronary anastomoses freely at arteriolar level, between the inter-ventricular arteries. If the intraventricular arteries meet at the apex, this provides maximum anastomoes. If the meeting place of the intraventricular arteries falls short of the apex above or below, this diminishes the potential anastomotic area. In 10% of the individuals the inferior as well as the anterior interventricular artery is a branch of the left coronary, in these cases there is no anastomoses between the coronaries.

Potential anastomoses exists between the coronary arteries and pericardial arteries which are derived from the pericardiophrenic, the bronchial and the internal thoracic arteries. In very rare instances one of these may open to replace a coronary artery.

DISTIBUTION OF CORONARIES

Right ventricle is supplied by the right coronary artery except at the upper margin of its anterior surface, where it is supplied by branches of anterior interventricular arteries. Left ventricle is supplied by the left coronary artery except for a narrow strip of the diaphragmatic surface where it is supplied by the inferior

interventricular artery. The two interventricular arteries share the supply of the interventricular septum, usually about equally.

The anterior surface of the right atrium is supplied by right coronary artery. The posterior surface and the auricular appendage of the left atrium are supplied from left coronary artery.

SA Node: It is supplied by a branch of right coronary artery in 60% of cases and from left coronary artery in 40%. AV node and bundle of His are supplied by the inferior interventricular artery, which arises in 90% of cases from the right coronary and in only 10% from the left coronary.

Dominant Arteries: In 67% of the cases right coronary is dominant, 15% of cases left coronary and in 18% of cases there is a balanced coronary arterial pattern.

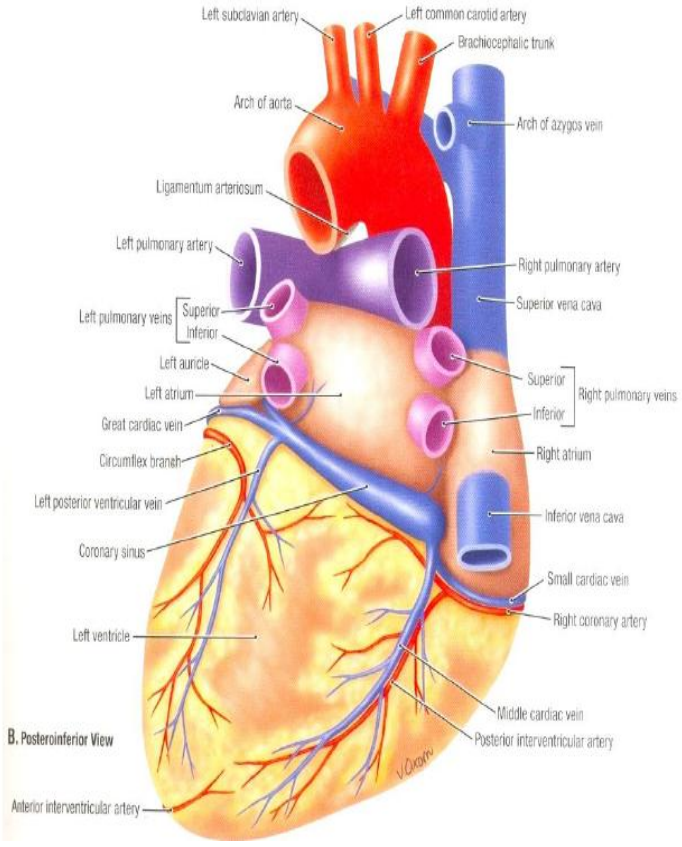
PHYSIOLOGY OF CORONARY CIRCULATION

Functionally, the right and left coronary arteries behave as end arteries, although anatomically there are numerous intercoronary anastomoses in most of the normal hearts in the order of 40% microns in diameter. Only the inner 75-100 microns of the endocardium can obtain significant amount of nutrition directly from the blood in the cardiac chamber.

HEART AND VESSELS

Figure C

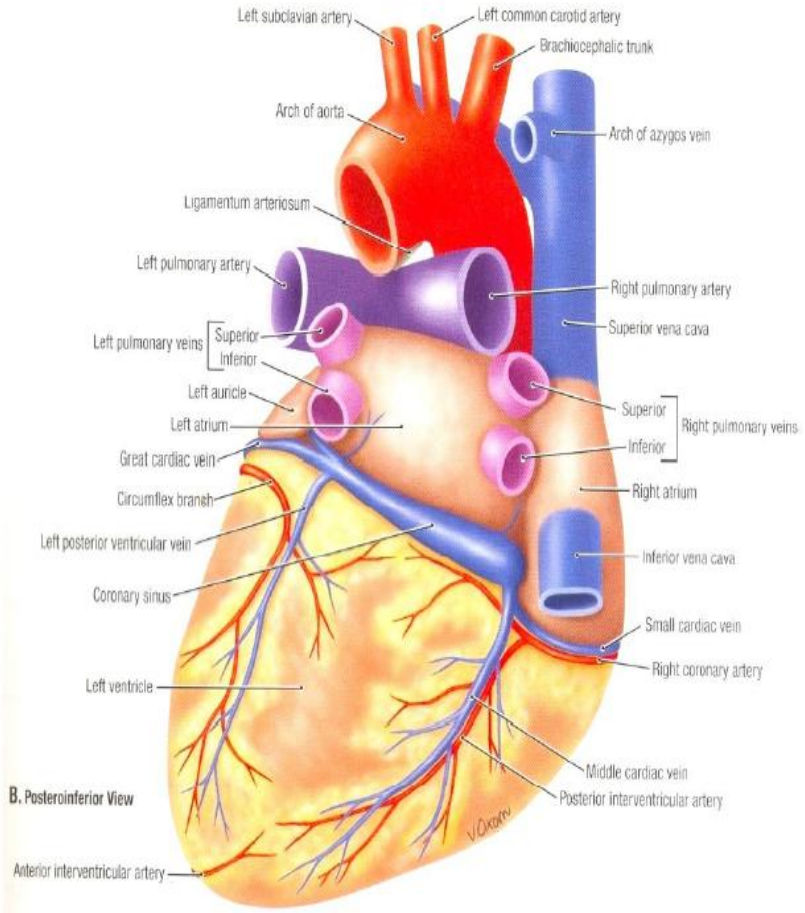
The Heart is the main pumping organ of the body .It has to give its pumped blood to whole body to enable adequate oxygen supply to the body. This occurs through an arterial tree, and the main vessel in that arterial tree is Aorta. This vessel arises from left part of the ventricles of heart and carries blood rich in oxygen.



HEART AND VESSELS

Figure D

The ascending aorta arises from the left part of the ventricle of heart at the level of 3rd intercostals space of the chest.The part of the mediastinum which contains heart and vessels of the heart is the middle mediastinum.The wall of the great vessel aorta has 3 dilatations which are called as aortic sinuses.



NORMAL CORONARY FLOW

Resting coronary blood flow in human beings averages about 225 ml/ min or 0.7-0.8 ml/G of heart muscle or 4.5 percent of the total cardiac output. Four to five fold increase can occur during exercise.

PHASIC CHANGES IN CORONARY BLOOD FLOW

As a result of cardiac muscle compression blood flow decreases duringsystole and increases during diastole, in which left ventricle is more affected than the right because of its thickness.

The intramyocardial pressures compress the subendocardial blood vessels more than the outer vessels, which throttle its blood supply and to compensate for this one, the subendocardial vessel are much larger than the nutrient arteries in the middle and outer layers of the heart, which increases the blood flow during diastole proportionately.

MAGNESIUM DEFICIENCY

Magnesium deficiency is becoming more commonly recognized due to increased clinical awareness, and the greater frequency of assessment of magnesium status by a physician. The incidence of magnesium deficiency in hospitals , which are tertiary centres is usually 10% , but it rises to 60% in intensive coronary care units. The main symptomatic causes of hypomagnesemia is most commonly due to GI loss or renal loss of magnesium The main etiologies of magnesium loss are:

DRUGS

-Diuretics

-Frusemide, ethacrymic acid)

-Aminoglycosides

-Cisplastin

-Cyclosporin

-Amphotericin-B

• **Metabolic acidosis**

-Starvation

-Ketoacidosis

G I disorders

- Continued NG suction
- Poor GI Absorption
- Severe Bowel resection
- Loose stools, Acute or chronic
- PEM
- Catastrophic pancreatitis
- Decreased serum magnesium in Neonates

Magnesium loss via Kidneys

- Continued iv fluid therapy.
- Loss due to osmotic factors
- Increased serum calcium
- Other Diseases of kidney like.

  Chronic pyelonephritis , interstitial nephritis and glomerulonephritis

  Diuresis period of ATN

Renal tubular acidosis

Post renal transplantation.

Hypomagnesemia may be encountered in about 28% of patients with acute hemorrhagic or edematous pancreatitis. The low serum magnesium concentration may be due disorder predisposing to the pancreatitis.

RENAL MAGNESIUM WASTING

Magnesium loss can occur through many route, but the most main route is kidney route for magnesium loss.The renal absorption of the magnesium, the main site is proximal tubule of the nephron and is dependant on Na reabsorption[63].So, continous therapy with sodium containing iv fluids will lead to mg loss. Hypercalcemia is shown to decrease magnesium absorption in the proximal tubule and loop of henle and is perhaps the mechanism of renal magnesium wasting or the tendency towards hypomagnesemia in most hypercalcemia states[64].

Gastrointestinal Disorder

The content of magnesium in the stools and biliary fluid is greater compared to other fluids, so hypomagnesemia is common in patients with chrons disease and intestinal inflammation. Vomiting and loose stools form an important component as magnesium because of the high Mg content of GI tract. The damage to the intestinal mucosal layer cells due to the therapeutic interventions for many malabsorption states like whipples disease can lead to low serum magnesium levels. Another cause of low serum

magnesium levels is because of poor gastrointestinal absorption of lipids leading to poor magnesium absorption. Various surgical interventions for obesity and poor GI absorption like Intestinal resection can also result in low serum magnesium[65].

The commonest cause of magnesium wasting is concurrent use of diuretics. Diuretics acting on the proximal tubules, such as carbonic anhydrase inhibitors and osmotic agents, may increase moderately magnesium excretion. Diuretics acting at the loop of henle such as frusemide and ethacrynic acid result in magnesium deficiency. Aminoglycoside therapy initially with capreomycin, gentamycin and recently with tobramycin, amikacin has produced renal magnesium wasting. Another cause of magnesium loss and hypomagnesemia is Therapy with Amphotericin B. Another chemotherapeutic drug Cisplatin alo causes loss of Mg through kidney route in many patients treated with patients.

Endocrine and Metabolic Disorders

Many diseases associated with various systems in body are associated with low magnesium levels. Of these an important diseases involve the hormonal system which controls the metabolic functions of the body. Many Of the diseases like Type 2 and type 1 diabetes mellitus is associated with low serum levels of magnesium[68]. The mechanisms responsible may be varied. These include the effect of the hormone insulin on the distribution of Mg in bodyfluids. It drives Mg inside the cells. Also loss of magnesium may occur through the kidney route and this accounts to the role of osmotic factors[69]. Hypomagnesemia can be found in association with a number of other

endocrine abnormalities. Phosphate depletion has been shown to result in urinary magnesium wasting and hypomagnesemia. That is hypophosphatemia is a contributing factor in the development of magnesium deficiency. Other conditions leading to urinary magnesium wasting and hypomagnesemia is thyrotoxicosis. The cause of Mg deficiency associated with disorders involving Renin Angiotensin aldosterone system may be related to the increase in the human body fluid status and the consequent excretion of mg through the kidney route.

Miscellaneous Causes

Mg depletion may occur because of unusual routes of excretion of Magnesium such as increased sweating and other rare routes of excretion.

EPIDEMILOGY OF CORONARY HEART DISEASE

Coronary Artery Disease (CAD) is a major cause of morbidity and mortality in individual aged 45 years or more throughout the world including India. Wide variations have been reported in the prevalence rate of CAD in different geographical regions. Finland and US lead all other countries in death rates from CAD.

In the US it is estimated that for those over 30 years of age 213 per 100,000 individual have ischaemic heart disease. Accurate data regarding the prevalence of CAD in India are not available. Surveys carried out in recent years, in different geographical locations and in small population groups using different protocols, estimate a prevalence rate of about 5% in urban population and a much lower prevalence in the rural setting.

The pattern of CAD in India has been reported to be as follows:

a) Males are affected more than females.

b) Hypertension and diabetes accounts 40% of all cases.

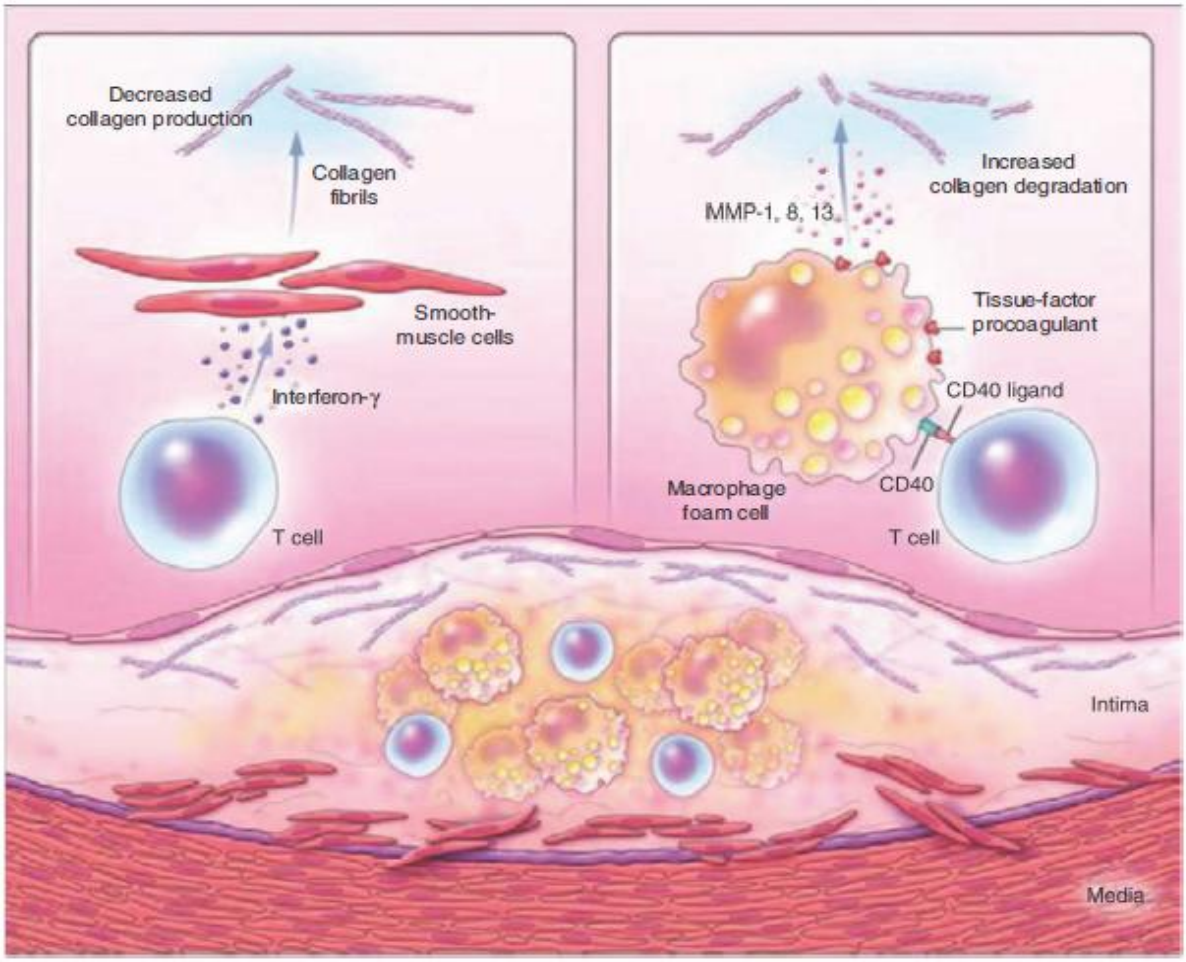
c) Heavy smoking is responsible etiology in a good number of cases.

d) Other factor includes high fat & energy rich diet, sedentary life style.

PATHOGENESIS OF ATHEROSCLEROSIS

Figure E

The lesion which occurs at the onset of atherosclerosis is Fatty streak which is formed by the accumulation of fatty compounds on the vessel wall in the intimal layer of blood vessel.Then the migration of wbcs to the wall of the heart vessels occurs and these cells get loaded with lipid and forms the so called foam cells.



PATHOPHYSIOLOGY OF ACUTE MYOCARDIAL INFARCTION

Myocardial infarction generally occurs when there is an abrupt decrease in coronary blood flow following a thrombotic occlusion of a coronary artery previously narrowed by atherosclerotic plaque fissures, ruptures or ulcerates and when conditions favor thrombogenesis takes place, so that a mural thrombus forms at the site of rupture and leads to coronary artery occlusion. After an initial platelet monolayer forms at the site of the ruptured plaque, a variety of agonists (collagen, ADP, epinephrine, serotonin) promote platelet activation. Following agonist stimulation, there is production and release of thromboxane A2 , further platelet activation and potential resistance to thrombolysis.

In addition to generation of thromboxane A2 , activation of platelets by agonists promotes a conformational change in the glycoprotein IIb, IIIa receptors. Once converted to its functional state this receptor develops a high affinity for the sequence arginine – glycine – aspartic acid on the fibrinogen alpha chain and also for a dodecapeptide sequence on the fibrinogen gamma chain. Since fibrinogen is a multivalent molecule, it can bind to two different platelets simultaneously, resulting in platelet cross linking and aggregation.

Ultimately the amount of myocardial damage caused by coronary occlusion depends on the territory supplied by the affected vessel, whether or

not the vessel becomes totally occluded, native factors that can produce early spontaneous lysis of the occlusive thrombus, the quantity of blood supplied by collateral vessels to the affected tissue, and the demand for oxygen of the myocardium whose blood supply has been suddenly limited.Depending on the extent of coronary occlusion, ischemia can be limited to the subendocardium or can involve the entire thickness of the myocardium, ie, a transmural infarction.Transmural infarction typically results in ST elevation myocardial infarction where as subendocardial ischemia leads to ST depression in ECG and is termed as Non- ST elevatin myocardial infarction.

CLINICAL FEATURES OF ACUTE MYOCARDIAL INFARCTION

Acute myocardial infarction presents itself as a sudden catastrophic incident and its definite clinical picture may be established without warning.

The clinical pictures can be classified as follows:

1) Cases dominated by chest pain
2) Cases dominated by shock
3) Cases dominated by pulmonary edema or other evidence of LV failure.
4) Cases characterized by the gradual development of CCF
5) Cases dominated by complication
6) Some cases may present with combination of any of the above.

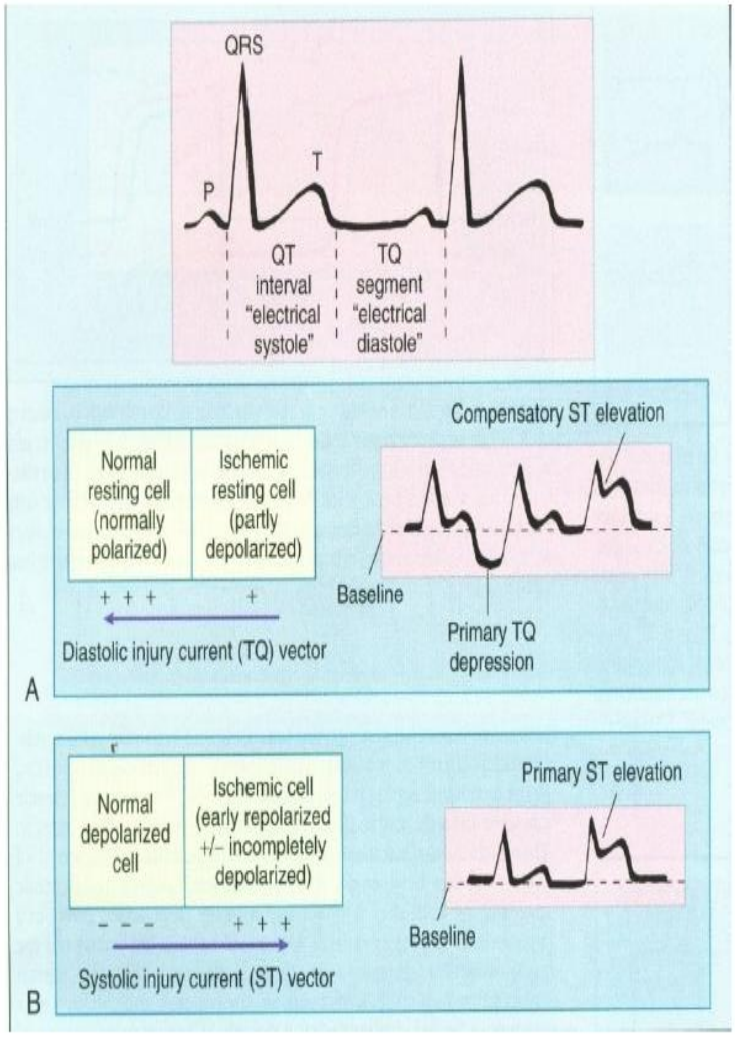
1. CHEST PAIN

In 80-85% of cases this is a presenting complaint. It is a deep visceral pain, involving the central portion of the chest and epigastrium, described as tightness, heaviness or constriction in the chest. In 25% of cases, it radiates to the arms and commonly it is often accompanied by weakness, sweating, nausea, giddiness and anxiety. It may occurs during exertion and emotional outbursts, not relieved with rest and makes the patient to move about in an attempt to find a comfortable position.

Figure F

PATHOPHYSIOLOGY OF ISCHEMIC ST ELEVATION

The pathophysiology of myocardial infarction involves thrombosis nof the lumen of the blood vessels leading to ischemia of myocardium leading to myocardial infarction. This can be ST elevation or NSTEMI. ST elevation myocardial infarction has a pathogenesis of total block of coronaries leading to full thickness destruction of myocardium.



2. Breathlessness

Second most important symptom, breathlessness may be sudden in onset and intense or it may be exertional. It is common in those who had painless myocardial infarction' particularly diabetics and aged individuals, and those having complications like cardiogenic shock and pulmonary edema.

3. Sudden loss of consciousness, a confusional state, a sense of profound weakness or unexplained fall in blood pressure with giddiness, syncope and/ or convulsions may be a presenting complaint.

4. Choking sensation in the neck may be the only presenting symptom.

5. Some patients present with gradual onset of breathlessness, paroxysmal nocturnal dyspnea and pain in abdomen with oliguria and swelling of lower limbs, a picture that of CCF.

6. In rare cases the infarct may go unrecognized until endocardial thrombosis resulting from its leads to systemic embolism.

Physical Signs. Patient may come with the hand on their pericardium indicating the site of maximum intensity of pain (Levine sign). often associated with perspiration and coolness of extremities, cyanosis may be there when the patient is having severe pulmonary edema or shock.

Pulse

May show bradycardia, normal sinus thythm, tachycardia with or without irregularities, depending upon the presence or absence of arrhythmias and the type of arrhythmia.

Blood Pressure

Usually shows an initial rise because of pain, anxiety or the unfamiliarity of the environment, which will become normal within 3 or 4 days. Fall in the blood pressure may be due to cardiogenic shock or due to 'Bezold-Jarisch reflex', which is due to increased vagal tone that occurs in inferior wall infarction.

Neck veins:

Collapse of neck veins occurs when patient is in shock, cannon waves can be made out in complete heart block in which they are irregular.

Precordium

The apical impulse may be difficult to palpate. In about one-fourth of the patients with anterior wall infarction, an abnormal systolic pulsation, develops in the periapical area within the first few days of illness, which may resolve later, which represents a transient, palpable systolic bulging of the infarcted ventricle. Other physical signs of ventricular dysfunction that may be present are, muffled heart sounds, atrial (S4) and ventricular (S3) gallop sounds and paradoxical splitting of the

2nd sound. A transient apical systolic murmur due to mitral regurgitation secondary to papillary muscle dysfunction during acute infarction may occur. A pericardial friction rub is audible if infarction is transmural in most of the cases. Temperature elevations in the range of 37 to 38ºC are common during the first 3 to 4 days due to myocardial necrosis.

Respiratory System: Tachypnea is common and crepitations are heard at the base or all over the lung fields depending upon the amount of pulmonary congestion.

Gastrointestinal System: enlarged tender liver will be present when patient is in CCF.

Central Nervous System: Anxiety, restlessness, stupor, coma, focal neurological deficit may occur when the patient is having fall in blood pressure and/ or thromboembolic phenomenon.

Renal System: Oliguria may be present if the patient is having fall in blood pressure.

MAGNESIUM AND ACUTE MYOCARDIAL INFARCTION

Epidemiological studies have suggested that the incidence of myocardial infarction and of sudden death is higher in areas of soft water intake . Cardiac magnesium content has been reported to be low in patients whose death was attributed to myocardial infarction[16]. It is unknown however, if the low cardiac content proceeds the myocardial infarction or is result of it. Cardiac magnesium exchanges quite rapidly with plasma magnesium and a number of clinical studies have shown a fall in the serum magnesium concentration within the first 24 to 48 hours after myocardial infarction. Infarcted myocardium has been repeatedly shown to have reduced magnesium content. But the results regarding the study of serum magnesium values in first 24 hours, following acute myocardial infarction has been variable. Some found no significant change of serum magnesium. It has, therefore been proposed that serum magnesium has an inverse relationship with coagulability of blood and serum cholesterol levels, following acute myocardial infarction.

Myocardial injury was established by histological examination of cardiac tissue. A significant rise in urinary magnesium excretion was observed during the first two hours after which the level declined but was still maintained above the control level significantly in infarcted myocardium. Magnesium content decreased significantly in infarcted myocardium.

MANAGEMENT OF STEMI

Figure G

Management of Patients with Acute myocardial infarction is dependant on various factors. Of these the most important factor is Time. ST elevation Myocardial infarction can be treated with two options.One is percutaneous coronary intervention and the other option is Thrombolysis using fibrinolytics.

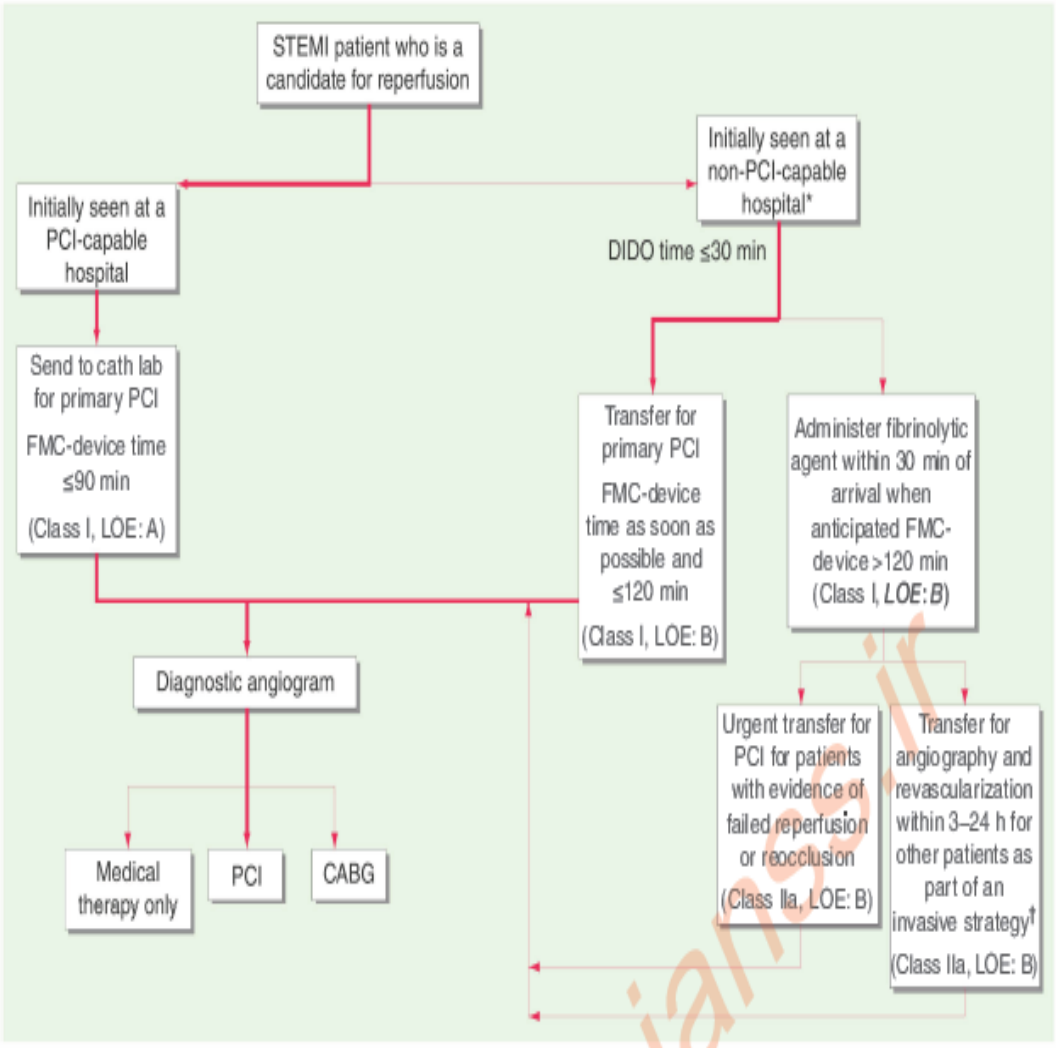


FIGURE G

Various authors have reported a decrease of serum magnesium following MI. Abraham S et al[17] (1980) from Israel checked serum magnesium levels of forty two patients of acute MI, nine patients of coronary insufficiency and fourteen patients of non-cardiac chest pain. Patients with acute myocardial infarction and those with acute coronary insufficiency had lower serum magnesium levels than eighty controls natural for age and sex, whereas there was no difference in patients with non-cardiac chest pain. There was a significant fall of serum magnesium during the first five days andth normal levels were reached by the 12 day.

Singh A et al[60] (1976) checked serum magnesium levels of twenty patients of acute MI on the first 7 and 12 day of admission. In all the cases, there was a significant fall of serum magnesium on the first day.

Babel S.Bhatnagar, HNS Bhatnagar[18] (1983) from Rajasthan tried to determine the prognostic significance of serum magnesium levels in acute MI. Twenty five patients of acute myocardial infarction were studied. Serum magnesium was found to be significantly lowered on the first day and it gradually rose to normal value by the twenty first day.

But some studies have showed that magnesium depletion resulted in lowered intracellular magnesium and potassium, increased intracellular calcium and sodium, and focal cardiac necrosis[19] . Patients with coronary artery disease

have been found to have decreased amount of exchangeable magnesium and patients sustaining an acute myocardial infarction were found to retain abnormally high amounts of magnesium during magnesium tolerance testing and to have lower skeletal muscle magnesium content suggesting the presence of magnesium deficiency[20].Magnesium depletion predisposes to vascular spasms, including coronary artery spasm and potentiates the contractile response to pressor agents such as angiotensin II and norepinephrine. Magnesium depletion potentially could worsen angina and precipitate acute myocardial infarction. The effectiveness of magnesium therapy in acute MI has been reported to decrease infarction size, decrease the incidence of cardiac arrhythmias and lower mortality rate[21,22,23}.

Mechanism of Tachydyrarrhythmias:

The exact mechanism behind the effect of magnesium on cardiac dysarrhythmias is unknown. Magnesium is essential for activation of ATP, which maintains the sodium-potassium pump. Therefore, it may have an important role in maintaining the resting membrane potential of electrocardiac cells, which depend on the intracellular potassium gradient. Magnesium deficiency is associated with loss of intracellular potassium, an increase in intracellular sodium, and an increase in cell excitability. One theory of magnesium and dysarrhythmias suggest failure of potassium to reenter the depolarized cell or a diastolic leak of potassium from already depolarized cells may promote aberrant condition, reentry phenomena and ventricular fibrillation[26,27].

Another theory is that magnesium acts as a calcium blocking agent. The increased intracellular sodium linked with magnesium deficiency may be followed by a sodium-calcium exchange causing an increase in intracellular calcium. Phasic influx of intracellular calcium is linked with transient depolarization and repetitive dysarrhythmias, infusion of magnesium cause a clinical picture similar to that produced by infusion of a calcium blocking agent, peripheral vasodilatation, flushing, a decrease in blood pressure and a decrease in contractile strength of the heart[28].

Lysophosphatidyl choline (LPC) is an endogenous lipid released from cell membranes during ischemia and has potent local effects on cardiac tissue. LPC causes membrane depolarization by decreasing potassium conductance of the inward rectified current and induces cardiac arrhythmias. LPC also triggers the accumulation of intracellular calcium in heart cells by inhibiting the sodium-potassium adenosine triphosphatase (ATPase) pump[29] . Increased systolic calcium can be detrimental to cells by activating calcium dependent phospholipases and proteases and by generating additional toxic fatty acids.

Excess free intracellular calcium also potentates the harmful effects of free radicals [30] . Because magnesium is a critical cofacors of myocardial ion pumps and antagonizes calcium influx. Antiarrhythmic effects of magnesium during ischemia were mediated by inhibition of increasing intracellular calcium induced by LPC.

Ventricular Tachydysarrhythmias and magnesium

In 1935, Zwillinger [31] injected 15 ml of a 20% solution of magnesium sulfate ($MgSO_4$) as a bolus into left ventricle of a patient with ventricular fibrillation resistant to other therapy. In 1943, Boyd and Schesf [32] used 10-20 ml of IU 10% MgSO to treat spontaneous dysarrhythmias with an approximately 50% success rate. Rasmussen et al[33] , experimental patients received approximately 1200 mg of magnesium chloride ($MgCl$) in the first 24 hours after AMI and approximately 300 mg in the second 24 hours. Results were compared with placebo control group. Those treated with $MgCl$ had significantly fewer incidence of dysarrhythmias requiring intervention (21% versus 47%) ($p<0.05$). Ventricular dysarrhythmias induced by digitalis toxicity are extremely responsive to magnesium therapy. Hypomagnesemia is common during digitalis toxicity and even in the presence of normal serum magnesium, intracellular magnesium is frequently low. Magnesium counteracts the inhibitory effects of digitalis on sodium/ potassium ATP. During digitalis therapy, there is an increase in intracellular calcium leading to augmentation of ionotropism and excitability [34] . In a study in monkeys, low magnesium levels were associated with a decrease in tolerance to digitalis and the duration of digitalis toxicity was prolonged.

Holden et al[35] found a striking decrease in $Mg2+$ both during cardiovascular bypass surgery and 1-day postoperatively. Dysarrhythmias after cardiovascular surgery are believed to be partially caused by

hypomagnesemia resulting from the use of anticoagulants during surgery i.e., anticoagulants bind Mg . The use of Mg++ in the postoperative period has decreased the incidence of dysarrhythmias.

Torsades de Pointes

Torsades de Pointes (TdP) is a life-threatening ventricular dysrhythmia. This repetitive polymorphic ventricular tachycardia occurs in the presence of QT prolongation. TdP is most commonly induced by type Ia antidysrhythmic drugs such as quinidine or disopyramide. Other QT-prolonging drugs, such as amiodarone, have been reported to cause TdP. Hypokalemia and hypomagnesemia can potentiate the development of TdP and in rare cases can be the cause[36].In a longitudinal study of 12 patients with TdP treated with intravenous MgSO , a single bolus of 2g of MgSO completely abolished TdP within 5 minutes in nine patients[27] . In the other three patients, a second dose of MgSO was given 5 to 15 minutes later completely corrected TdP. No side effects were associated with the treatment. Similar findings were reported by Perticone et al[37].

Atrial dysrhythmias

Hypomagnesemia has been shown to make control of atrial fibrillation (AF) difficult [38] . In one study of 45 consecutive patients with symptomatic AF,20% had serum magnesium levels <1.5 mEq/L. In a blinded treatment protocol,

hypomagnesemic patients required twice the amount of IV digoxin to control AF. This study suggests that monitoring and replacement of Mg++ may be beneficial in patients with symptomatic AF, especially when digoxin therapy is considered[39].

MANIFESTATIONS OF MAGNESIUM DEFICIENCY SIGNS AND SYMPTOMS

The lack of Magnesium in diet in normal people leads to characteristic symptoms like change of character, spasticity of limbs,coarse tremor and muscle spasms.. The main causes of low serum magnesium levels are usually reactive to other major causes, so these causes may be masked by the manifestations of those primary causes. Manifestation associated with moderate to severe magnesium deficiency is shown in Table-1.

Table-1

Manifestations of Moderate to Severe Magnesium Deficiency

Biochemical

Hypokalemia

- Loss of potassium through urine.
- Intracellular Mg deficiency.

Hypocalcemia

- Decreased secretion of parathyroid hormone.
- Lack of effect of PTH on kidnys and bones
- Lack of response to vit D.

Neuromuscular

- Presence of carpopedal spasm
- Spontaneous Trousseau sign positivity.
- convulsions
- giddiness, aincoordination, visual disturbance, involuntary movements.
- skeletal weakness , atrophy and fibrillation
- Psychiatric manifestations

Cardiac Arrhythmias

ECG – PR and QT prolongations

AT,PAC,PVC

VT

VF

TdP

DIAGNOSIS OF MAGNESIUM DEFICIENCY

Serum Magnesium Concentration is affected b the compartmentalization of magnesium in various parts of the fluid compartments of the human body. The serum magnesium concentration may not reflect the intracellular magnesium content. The measurement of serum magnesium concentration is the most commonly employed test to assess magnesium status. The normal serum magnesium of less than 1.7 mg/dl usually indicates magnesium deficiency. Exogenous and endogenous catecholamines have been shown to result in a slight fall in the serum magnesium concentration and increased catecholamine secretion could be a contributing cause of hypomagnesemia in acute illness and stress[17] . Volume contraction and rhabdomyolysis (cellular magnesium release) can cause an increase in the serum magnesium concentration and may mask an intracellular magnesium deficit.

Other methods to estimate the physiologically active Mg has lead to the discovery of many new methods . Of these the most important is peripheral lymphocyte magnesium concentration which has a good relation to the concentration of physiologically active magnesium in various parts of body.

Magnesium Tolerance Test: This is among the commonest method used for estimating low serum magnesium level patients. Many studies have confirmed that the serum levels

of magnesium after a bolus dose of Mg is low in patients with low serum magnesium level as well as in those at risk for the same

The procedure is:

1. Obtain the initial level of the Mg – Cr(creatinine) value

2. Give 0.2 meq (= 2.4 mg) Mg / kilogram BW infused in fifty ml of 5% D over a period of few hrs.

3. Do a twenty four hour urine collection for Mg levels.

4. Obtain the amount of Mg left back in the body

5. Hypomagnesemia : the Criteria to diagnose

More than fifty percent retention = Hypomagnesemia definite diagnosis

More than twentyfive percent leftback = Hypomagnesemia probable diagnosis

Treatment of Magnesium Deficiency

Patient who lack magnesium or who manifest magnesium deficiency or are at risk for deficiency should be treated with magnesium containing diet, and is optimal in most patients. But those who lost Mg in loose stools or kindey loss of Mg should be treated with external doses of Mg. Kidney function tests should be checked in all patients presenting with low serum magnesium, Kidney is a major route of elimination of magnesium, ie more than 50 % is excreted in kidneys. So renal failure is a obstacle in

giving large intravenous doses of magnesium.Severe and symptomatic hypomagnesemia should be treated with parenteral therapy of Mg.

Administration of Parenteral Magnesium

Although a single dose of IV Mg^{2+} may be effective, the kidney will excrete a large amount of the dose delivered. The American Society of Hospital Pharmacists recommends that the maximum loading dose of Mg^{2+} be 150 mg/min . In emergency situations of ventricular tachycardia or ventricular fibrillation, the adult ACLS regimen is 1g diluted in 100 mL given over 1 to 2 minutes . During this treatment, the electrocardiogram should be monitored continuously to avoid cardiac toxicity. Blood pressure should be monitored closely during IV replacement of Mg^{++}, with the infusion slowed if the patient becomes symptomatically hypotensive. Common side effects of rapid IV administration of Mg^{++} include cutaneous flushing, sweating, and sensation of heat, hypotension, decreased deep tendon reflexes, somnolence, hypocalcemia, tetany, respiratory insufficiency , paralysis, and cardiac arrest can occur. These side effects can be alleviated by slowing the infusion. Patients also require monitoring of the serum magnesium level, neurologic status, respiratory status, and renal function. Patellar reflexes should be assessed before starting treatment and monitored carefully during treatment. Therapy should be stopped if the reflexes become suppressed.

If the patient has renal insufficiency, the magnesium dose is decreased

by 25% to 50% to prevent hypermagnesemia. Replacement therapy is closely guided by the serum magnesium level to avoid toxicity. A potential complication from MgSO4 administration is hypocalcemia. The sulfate binds with calcium, forming sulfate and reducing ionized calcium. Zaloga and Charnow recommended IV magnesium chloride, rather than MgSO4 to avoid calcium sulphate precipitation. Calcium gluconate should be kept available as an emergency treatment in the event of hypocalcemia, tetany or overdose apnea.

Administration of Oral Magnesium

For non-serious conditions oral forms of magnesium supplementation are available, although gastrointestinal absorption may vary. Large doses of oral magnesium salts often cause diarrhea. However, Mg++ in the Mg++ chloride salt form or in enteric-coated tablets is usually well tolerated. A course of three magnesium chloride tablets a day for 30 days will reduce the deficit for most patients . For patients taking diuretics, the substitution or addition of a potassium/ magnesium-sparing diuretic is probably beneficial.

HYPERMAGNESEMIA

Hypermagnesemia is usually because of treatment with Mg and can also be due to kdiney problem, which can be acute or chronic. The most common cause isn iatrogenic Mg supplementation which may be oral or parenteral administration. Mg level increases in response to GFR rate decrease but in early kidney disease serum Mg levels usually remain normal. Magnesium excess can be treated using treatment options like dialysis, but these procedures can result in high serum magnesium because of high concentration of magnesium in iv fluids and dialysate fluid.

Other causes of hypomagnesemia can be complications in pregnancy like treatment for eclampsia in pregnancy like magnesium sulphate therapy, addisons disease or decrease thyroid hormone levels.

Magnesium containing drugs like laxatives cause hypermagnesemia in patients with normal renal function.The central nervous system findings in patients with hypermagnesemia include loss of DTR. In patients with more profound increase of serum magnesium itn can lead to paralysis of skeletal muscles and lead to weakness of all 4 limbs, neck muscle weakness ,paraparesis ,apnoea or respiratory failure.But mentation may remain normal in patients at this stage of increased serum magnesium, but severe deficiency will lead to stupor and coma and even death.ECG changes of hypo

magnesemia include prolongation of PR interval,ventricular arrhythmias,supraventricular arrhythmias,intraventricular conduction problems.At very severe hypermagnesemia may lead to death from cardiac arrest. Magnesium can be used as a tocolytic and can be used in the treatment of preeclampsia and seizures associated with pregnancy and the basis for this action is direct depressant effect on neuromuscular junction,smooth muscles. Migrain treatment also employs Mg and this treatment is becoming popular. High dose of magnesium present in therapeutic doses of magnesium containing drugs can lead to hypermagnesemia in patients with abnormal renal function.Symptomatic cases can be treated with dialysis to remove the excess magnesium.

MATERIALS AND METHODS

MATERIALS AND METHODS

DATA SOURCE

60 Cases of Acute Myocardial Infarction , admitted to Intensive Coronary Care Unit of Thanjavur Medical College Hospital over 11 months ie., between July 2014 to May 2015.

INCLUSION CRITERIA FOR PATIENTS

Patients were diagnosed to have Acute Myocardial Infarction , only if they had 2 of the following characteristics:

1) Chest Discomfort.

2) ECG features of Acute Myocardial Infarction.

3) Elevation of Cardiac Enzymes.

Only those patients presenting to the hospital within 12 hours of the onset of symptoms were included in the study.

EXCLUSION CRITERIA FOR PATIENTS

Patients with hypokalemia.

Selected patients were subjected to detailed history and thorough physical examination and routine investigations like hemoglobin, Total leucocyte count, Urine examination,

blood sugar, Blood urea, Serum creatinine, serum electrolytes , fasting lipid profile, cardiac enzymes and Echocardiography was done in all cases.

Serum Magnesium level was done on Day-1 and Day-5.

METHOD OF SERUM MAGNESIUM ESTIMATION:

Method: Calorimetric end point test.

Reagent : Xylidyl blue reagent.

Magnesium Standard: 2.5 mg/dl.

Principle:

At alkaline PH , Magnesium reacts with Xylidyl blue and produces a chelating red compound.The increase in red colour or decrease in blue colour is proportional to magnesium concentration.

SPECIMEN

Non hemolysed sserum or Lithium Heparin plasma may be analysed since the magnesium concentration inside erythrocytes is 10 times greater than that in ECF, Hemolysis should be avoided and serum should be separated from the cell as soon as possible.

REFERENCE RANGE FOR MAGNESIUM

Serum Magnesium : 1.6 -2.4 mg/dl

RESULTS

RESULTS

Age Range(yrs)	Male	Female	TOTAL
30-40	8	1	9
40 – 50	9	5	14
50 – 60	10	5	15
60 – 70	14	4	18
70 - 80	4	-	4

TABLE 1 : Age and sex distribution of the study group

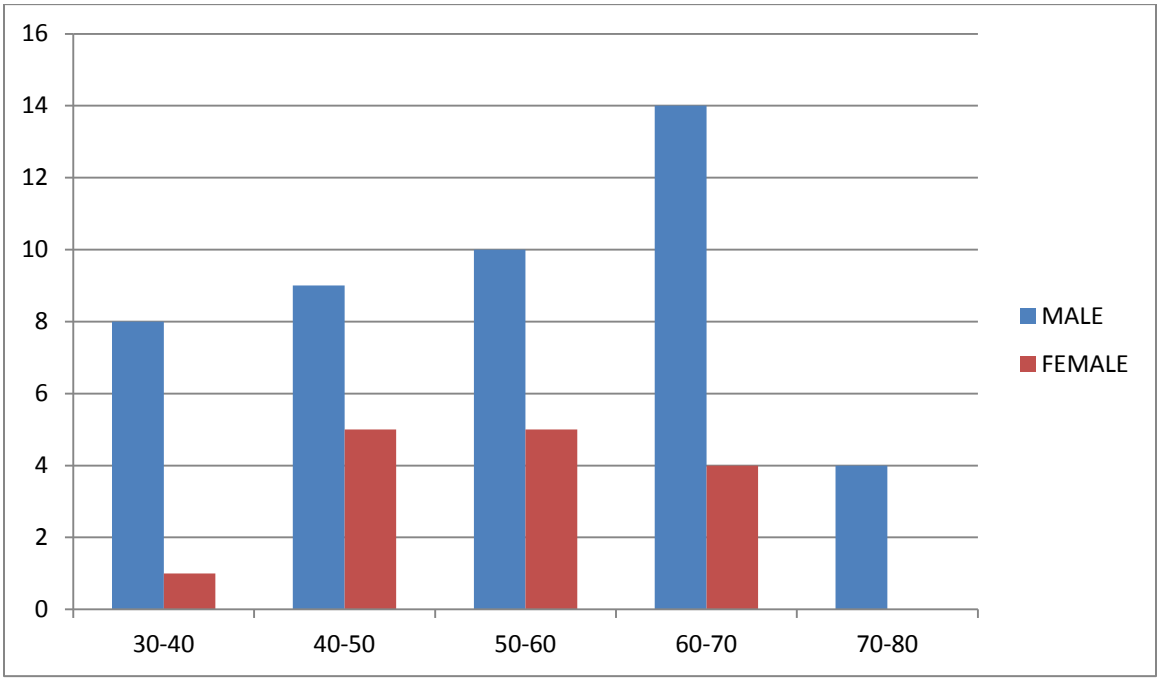


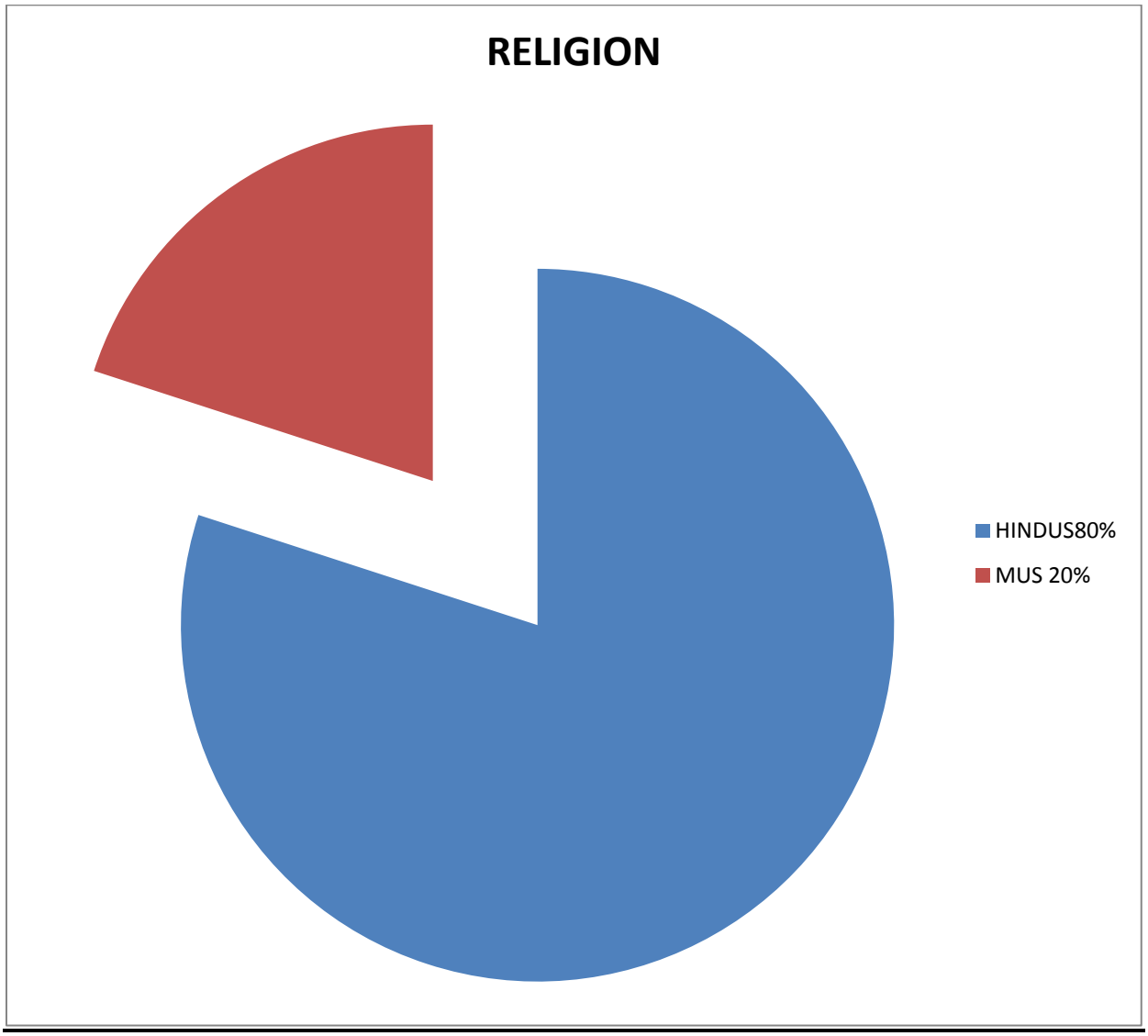
Figure 1 : Age and Sex distribution of study group

In this study of 60 cases , 48 were males and 12 were females with male female ratio of 4:1. The maximum incidence of acute myocardial infarction was seen between 6th and 7 th decades of life followed by fifth and sixth decades. 30 % of patients were in the 6 th to 7 th decades and 25 % of patients were in the fifth and sixth decades.

TABLE 2: Religion wise distribution of cases

RELIGION	NO. OF CASES	PERCENTAGE
HINDUS	48	80 %
MUSLIMS	12	20%

FIGURE 2: RELIGION WISE DISTRIBUTION OF CASES



DIET

In this study of 60 patients, 25 percentage (15 patients) were vegetarian by diet and 75 percentage (45 patients) of patients consumed mixed diet. Non – vegetarians have higher risk for acute myocardial infarction owing to the higher cholesterol content in the diet.

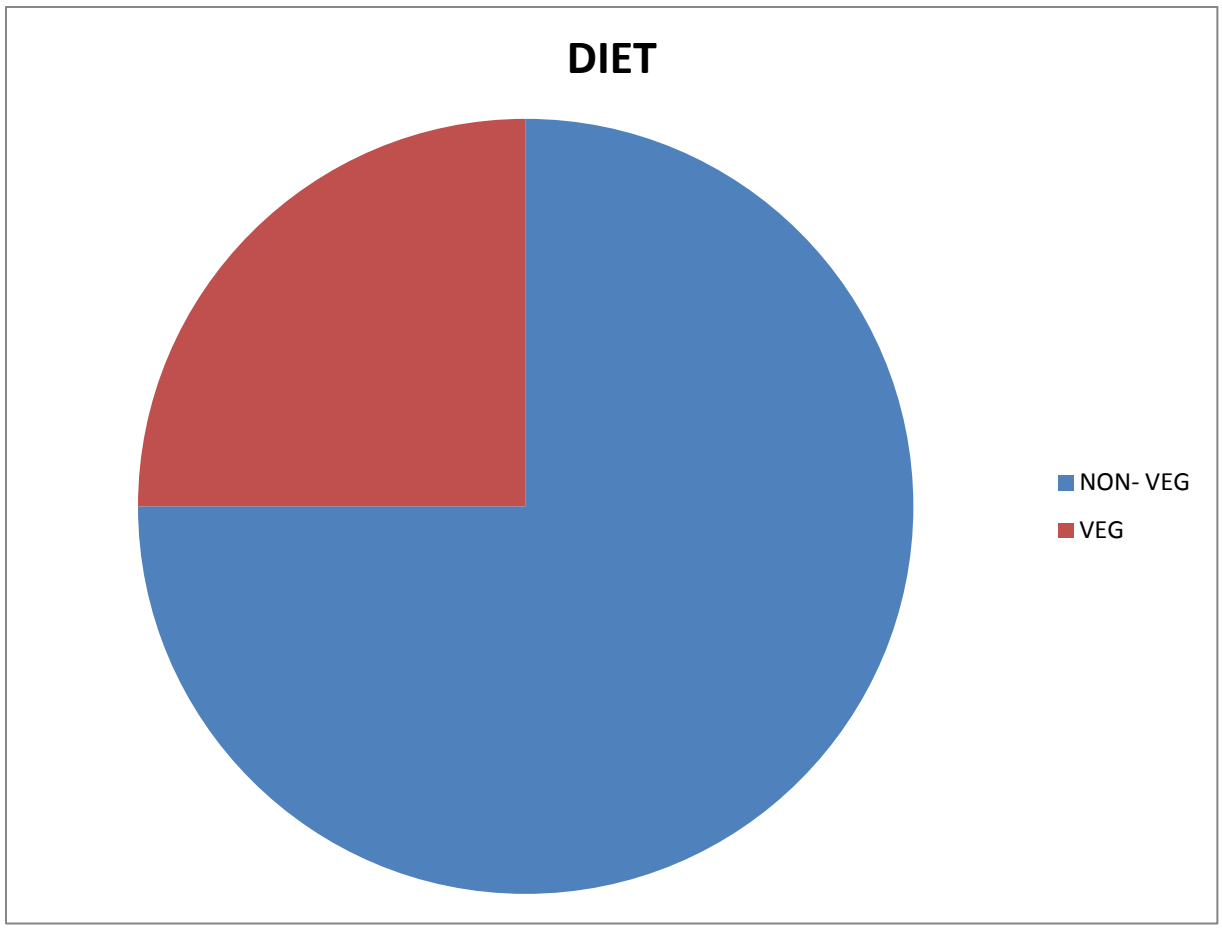


Figure 3

TABLE 3 : RISK FACTORS

RISK FACTORS	NO. OF CASES	PERCENTAGE
SMOKING	45	75%
FAMILY HISTORY OF HTN,IHD,DM,CVA	15	25%
OBESITY	12	20%
HYPERTENSION	30	50%
DIABETES MELLITUS	21	35%
DYSLIPIDEMIA	15	25%

SMOKING

In the study, smoking is the most common risk factor found in the patients with acute myocardial infarction. Cigarette smoking accelerates coronary atherosclerosis in both sexes and at all ages and increases the risk of thrombosis, plaque instability and myocardial infarction. In addition, by increasing myocardial oxygen needs and reducing oxygen supply, it aggravates angina.

HYPERTENSION

In the present study, out of 60 patients, 30 patients were found to be hypertensive based on history and blood pressure measure ment. In this study Hypertension was found to be the second main risk factor (50%) for the development of acute myocardial infarction.

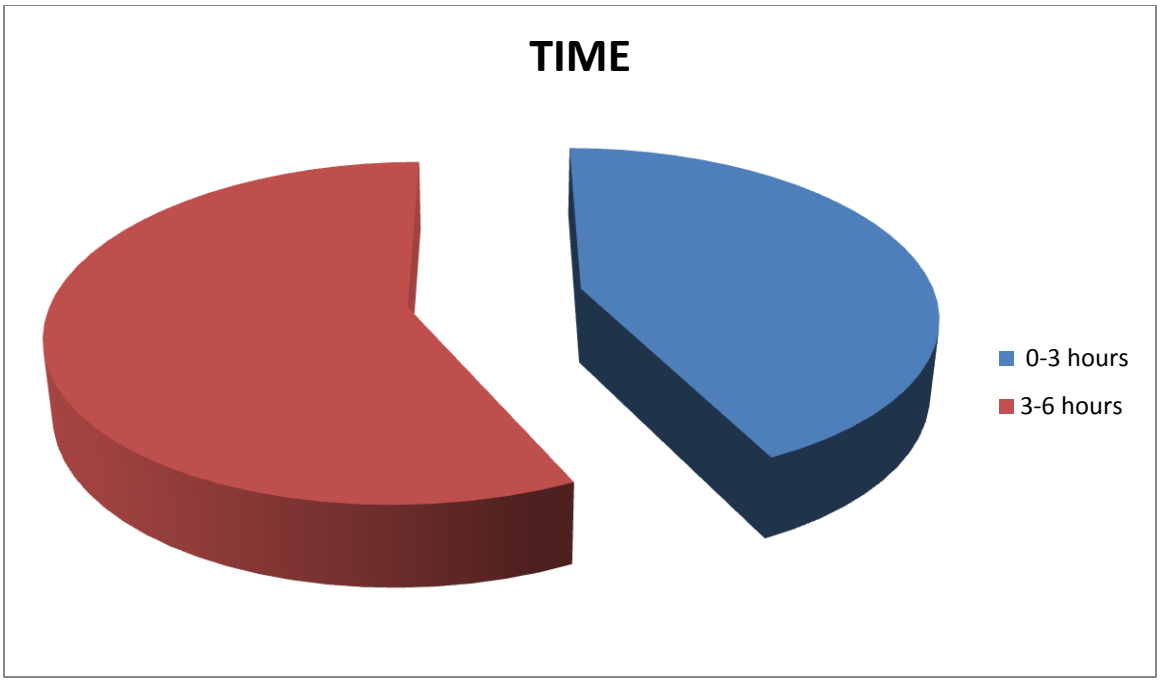
In the present study ,out of 60 patients, 21 patients (35%) were found to be diabetic and 15 patients (25%) were found to have dyslipidemia.

TABLE-4 : TIME OF PRESENTATION

TIME AT PRESENTATION	NO. OF CASES	PERCENTAGE
0 – 3 HOURS	15	25%
3 - 6 HOURS	30	50%

In the present study , 30 cases (50% of cases) presented to hospital between 3-6 hours of onset of chest pain and 15 cases (25%) cases presented between 0 – 3 hours.

Figure 4



PRESENTATION TO THE HOSPITAL

Presentation to the Hospital

Chest pain was the commonest symptom and was present in all of the patients in the present study (100%). In this study chest pain is associated with sweating 15 (25%) of patients. Chest pain is associated with breathlessness in 15 (25%) of the patients. Palpitation associated with chest pain was present in 6 patient (10%).

Variation in type of Myocardial Infarction

In the present study of 60 patients, 25 (41.66%) patients had anterior wall MI, 20 (33%) patients had inferior wall MI and 15 (25%) patients had anteroseptal MI

.

Serum magnesium in Acute Myocardial Infarction in Relation to Arrhythmia

In this cross sectional study of 60 patients, the mean serum magnesium level on day-1 in all 60 patients was 1.78±0.32 and the mean serum magnesium level on day-5 was 2.32±0.44.

Mean serum magnesium level in the group with Arrhythmia on Day-1 and Day-5

In the present study, out of 60 patients 30 patients had significant ventricular premature contractions/ ventricular tachycardia/ ventricular fibrillation during their 5-days course in the hospital

TABLE – 5: Serum magnesium levels in patients with Arrythmias

SERUM MAGNESIUM(mg/dl)	Day - 1	PERCENT	DAY - 5	PERCENT
< 1.6	12	20%	6	10%
1.6 to 2.50	18	30%	24	40%
>2.5	-	-	-	-

TABLE 6: Serum magnesium levels in patients without Arrythmias

Serum magnesium (mg/dl)	Day 1	PERCENT	DAY 5	PERCENT
<1.6	6	10%	-	-
1.6 – 2.5	18	30%	21	35%
>2.5	6	10%	9	15%

TABLE 7: MEAN SERUM MAGNESIUM LEVEL

	DAY 1	**DAY 5**
Mean serum Mg in 60 cases	1.78 +_ 0.32	2.32 +_ 0.44
Mean serum Mg level in patients with arrhythmia (30 patients)	1.58 +_ 0.30	1.96 +_0.32

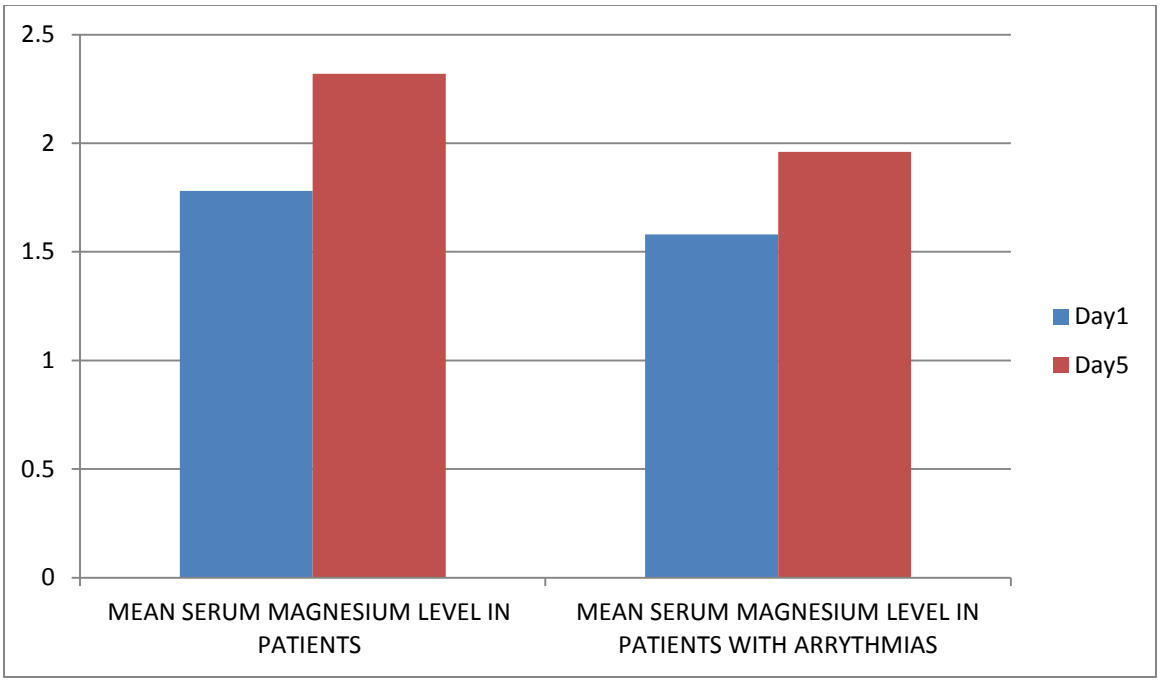


FIGURE -5 : MEAN SERUM MAGNESIUM LEVEL

	No of cases	Serum magnesium (Day 1)	p -value
Mean serum Mg in patients with arrythmia	30	1.58 +_ 0.30	<0.001
Mean serum mg level in patients without arrythmia	30	2.10 +_0.50	

TABLE: 8: Comparison of serum magnesium level in patients with Arrythmias and without Arrythmias (Day – 1)

The above table shows that out of 60 patients, 30 patients had arrhythmias. The mean value of serum magnesium on day-1 those with arrhythmias is 1.58±0.26 those without arrhythmias is 2.10±0.4 ($p<0.001$). There is a significant difference in the mangesium level in patient with arrhythmias and without arrhythmias.

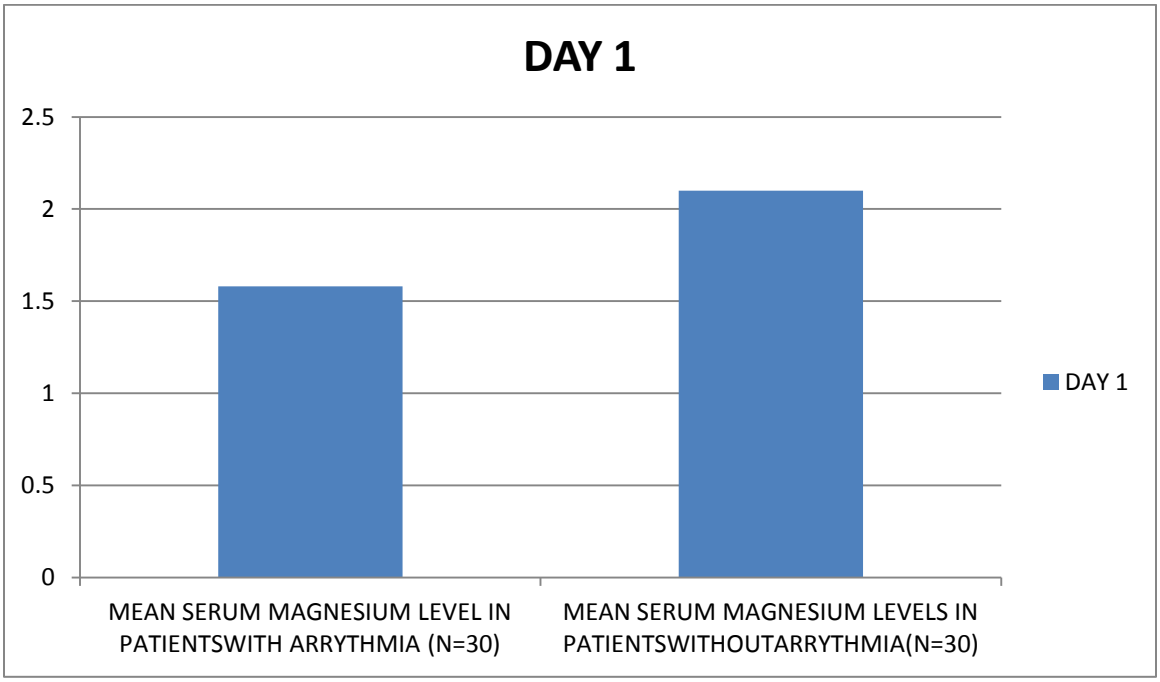
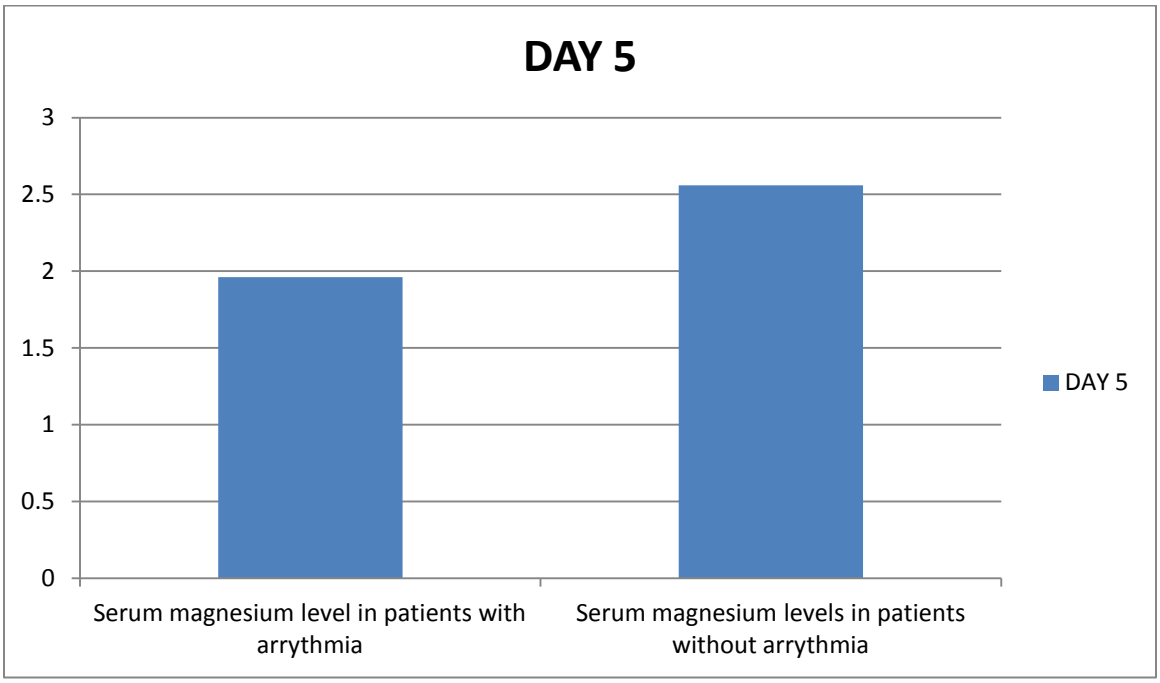


Figure 6

TABLE 9: COMPARISON OF SERUM MAGNESIUM LEVEL IN PATIENTS WITH ARRYTHMIAS AND WITHOUT ARRYTHMIAS (DAY – 5)

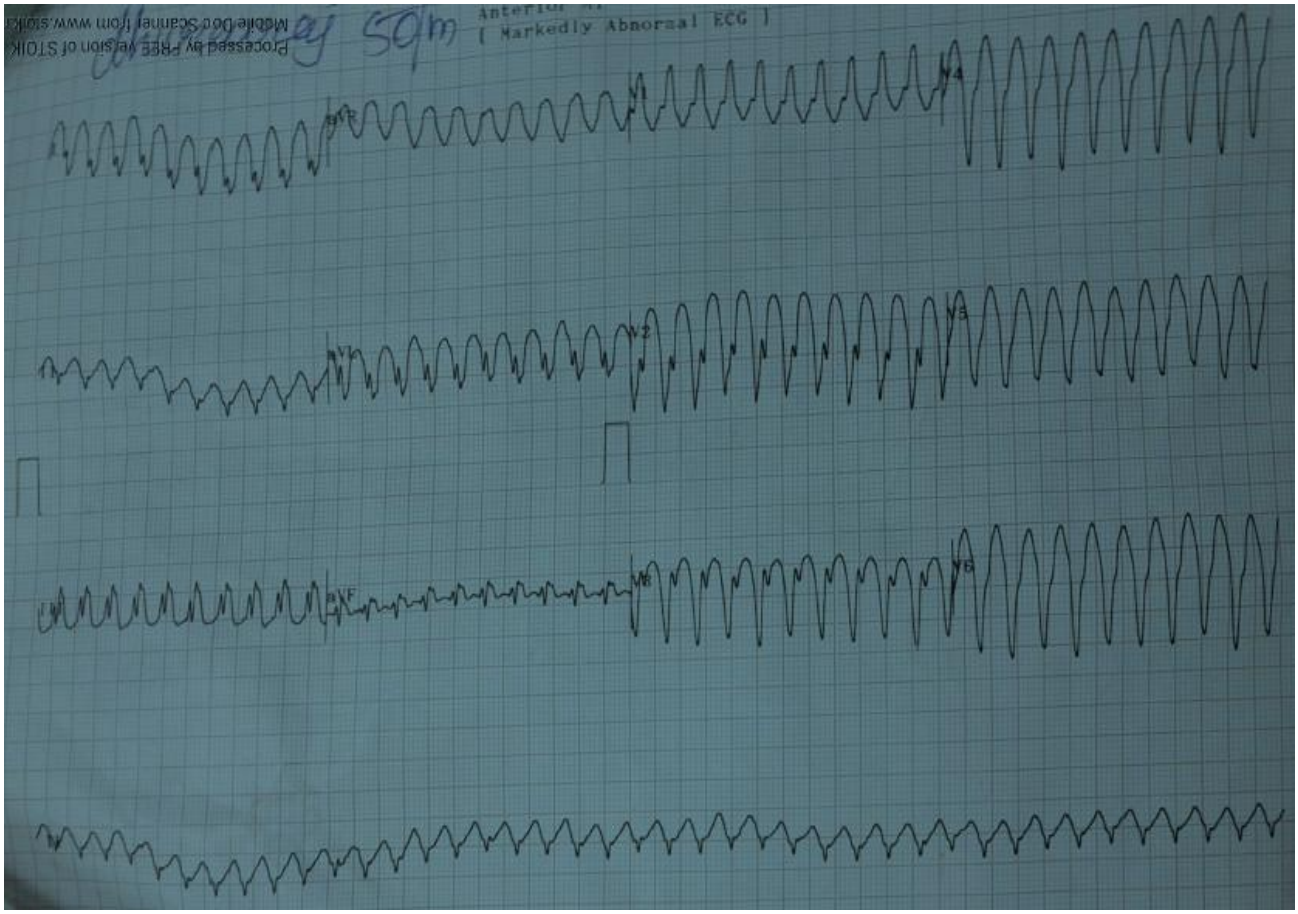
	no.of cases	**serum Mg Day-5**	**p- value**
Mean serum magnesium levels in patients with arrythmia	**30**	**1.96+_0.32**	**<0.001**
Mean serum Mg levels in patients without arrythmia	**30**	**2.56+_0.48**	

FIGURE : 7- Comparison of Serum magnesium levels in patients with Arrythmias and without arrhythmias (Day-5)



MORTALITY:

In the above study of 60 patients, 12 patients died during their 5 days hospital course. 8 patients were died of ventricular tachycardia or ventricular fibrillation, 4 patients died of cardiogenic shock. Mortality percentage was 20% .

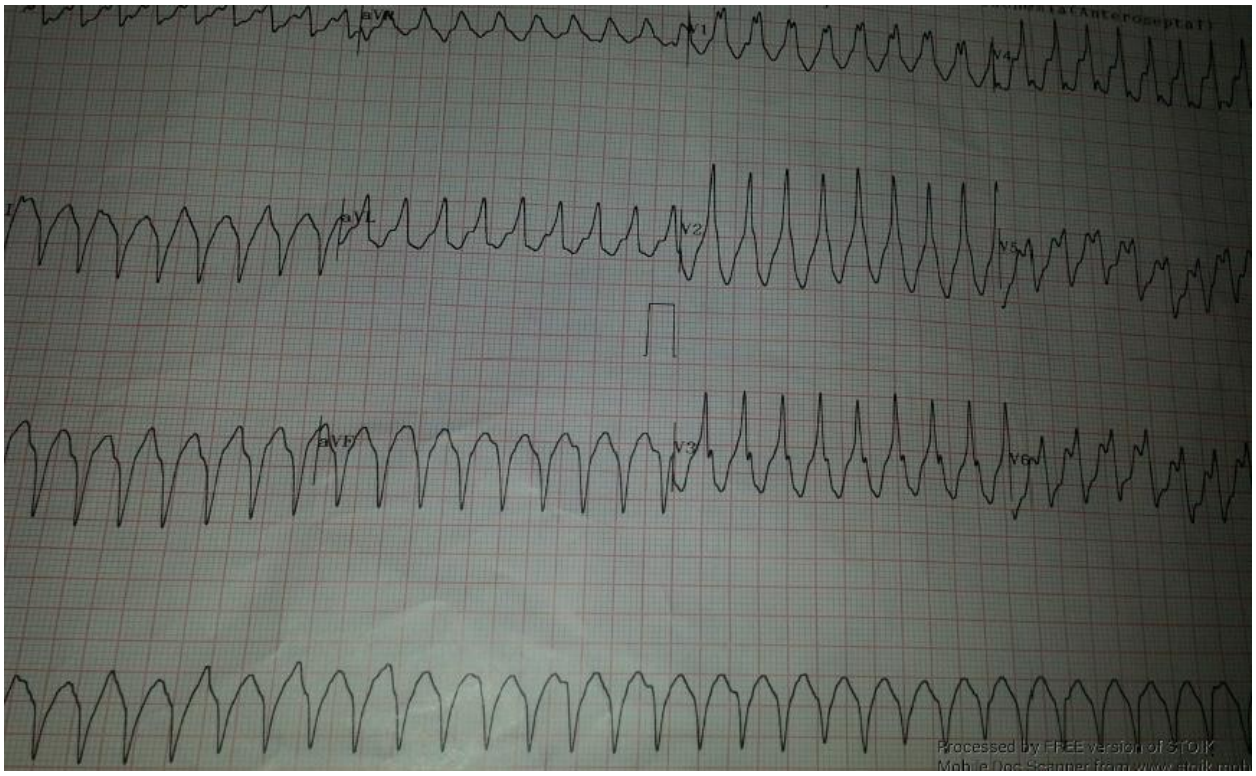


Mr.DURAIRAJ

50/M

29762

ACUTE EXTENSIVE ANTERIOR WALL MI

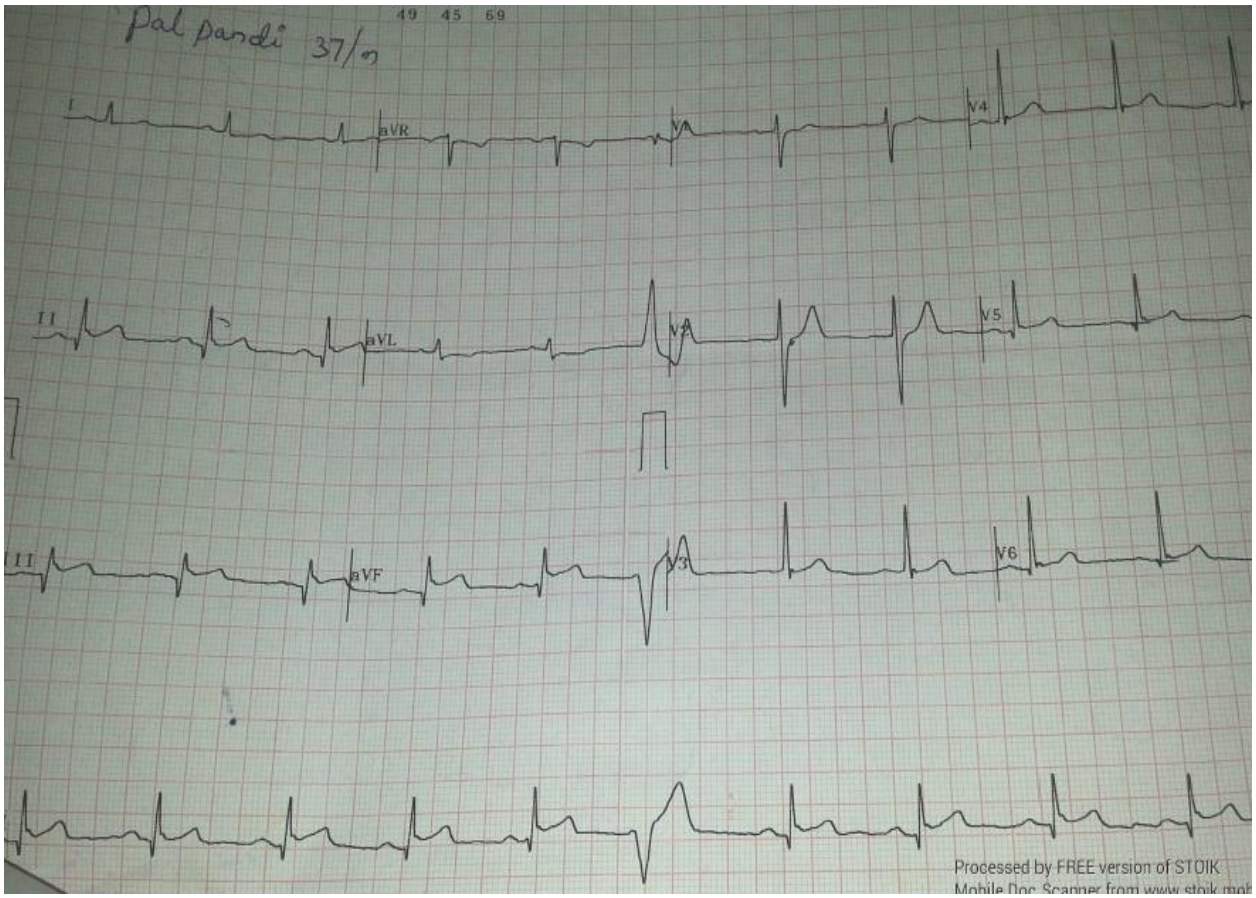


GANESAN

80/M

24824

ACUTE ANTEROSEPTAL MYOCARDIAL INFARCTION



PALPANDI

37/M

27729

ACUTE INFERIOR WALL MYOCARDIAL INFARCTION

DISCUSSION

DISCUSSION

Magnesium ion has emerged as a premier cardiovascular cation during the decade. It has been implicated in the pathogenesis of acute myocardial infarction and complication like arrhythmias. Magnesium is essential for activation of ATP, which maintains the sodium-potassium pump and also because of calcium blocking action magnesium has been implicated in relation to arrhythmias after acute myocardial infarction.

In the study group of 60 patients, 45 were males and 15 were females with a male-female ratio of 4:1. The maximum incidence of acute myocardial infarction was seen in the 6^{th} and 7th decades. In the present study of 60 patients, the mean serum magnesium level on day-1 in all 60 patiens was 1.78±0.32 and the mean serum magnesium level on day-5 was 2.32±0.44.

Abraham et al(13) reviewed magnesium level of 65 consecutive patients with an admission diagnosis of acute myocardial infarction. Serum magnesium concentration were low in patient who had AMI (mean 1.70 mg/dl, $p<0.001$) or acute coronary insufficiency (mean 1.61 mg/dl, $p<0.01$), but not in the control group or patients with non-cardiac chest pain (mean 1.91 mg/dl).

Singh A et al(60) checked serum magnesium levels of twenty patients of acute myocardial infarction on the 1st , 7th and 12th day of admission. In all the cases, there was a significant fall of serum magnesium on the first day. Dimtruk[63] in his series of 67 patients of ischemic heart disease showed a distinct reduction of plasma magnesium during the first 3 days following onset of disease, the level normalized by 15-25 days from onset of the disease.

Sachdev et al(64) (1978) in 30 patients of myocardial infarction determine the magnesiumlevels within 24 hours, 5th and 8th day and reportd as 1.83±0.087 mgm%, 1.91±0.149 and 1.97±0.089 as against control of 2.44±0.162 mgm%. The values were statistically lower on all the three days showing a progressive rise.

In the present study, the serum magnesium level on day-1 was significant lower in patients with arrhythmias than those without arrhythmia ($p<0.001$). There was an increase in serum magnesium from Day-1 to Day-5 in both those with arrhythmias and those without arrhythmias. Ceremuzynski et al (65) assigned 48 patients with acute myocardial infarction over 24 hours infusion of magnesium or placebo. The incidence of ventricular tachycardia (3 or more consecutive premature ventricular contraction at a rate faster than 120/ min) recorded by Holter monitoring was significantly reduced ($p<0.001$), but the incidence of other ventricular arrhythmias was not statistically different.

Raismusen et al(21) randomized 273 patiens with suspected acute myocardial infarction to intravenous magnesium or placebo. There is a significant decrease in the ventricular arrhythmia in the magnesium group compared to placebo ($p<0.05$). Shecter et al (66) randomized 103 patients with documented acute myocardial infarction to 48 hours infusion of magnesium or placebo. There is a significant decrease in mortality ($p<0.01$). There was also a non-significant decrease in the number of tachyarrhythmias requiring treatment (10/50) in the magnesium group compared to control (24/53).

Smith et al (67) randomized 400 patients with suspected AMI to a hours infusion of magnesium sulphate or placebo. Two hundred patients had confirmed acute myocardial infarction. The difference in mortality and incidence of ventricular dysarrhtymia requiring treatment between magnesium and placebo groups were not statistically singnificant.

Abraham et al[68] randomly assigned 94 patients with acute myocardial infarction to receive a daily magnesium bolus of 30 mmol or placebo for 3- days. There was no significant difference in mortality or lethal arrhythmias between patients treated with magnesium and those treated with placebo.

Felstedt et al(69) randomized 298 patients with suspected acute myocardial infarction to 24 hours infusion of magnesium or placebo. Myocardial infarction was documented in 162 patients. During the mean

observation period of 245 days, there was no difference in the incidence of tachyarrhythmias, magnesium infusion was associated with a significant increase in bradyarrhythmias. Singh et al[70] randomized 264 patients with suspected acute myocardial infarction to magnesium, potassium, 10% glucose or 2% glucose infusion. Myocardial infarction was confirmed in 228 patients. Mortality and ventricular tachycardia or fibrillation did not differ significantly between the magnesium group and placebo group.

Morton et al(71) randomized 76 patients to receive either magnesium infusion 0.38 mmol/l per kg every 12 hour or placebo over the first 36 hours of hospital, there was no difference in the incidence of ventricular tachycardia.

Dyckner T et al (7) during their 1½ years, 905 admission, 342 with acute myocardial infarction, 563 other diagnoses ere treated in the CCU on admission both acute myocardial infarction and non AMI group had significantly lower serum magnesium level than as reference group. The incidence of serious ventricular premature beats, ventricular tachycardia and ventricular fibrillation on admission was significantly higher in the hypomagnesemic patients with acute myocardial infarction.

CONCLUSION

CONCLUSION

This study was carried out in 60 patients of acute myocardial infarction who are admitted to the ICCU of THANJAVUR MEDICAL COLLEGE THANJAVUR.

1. The male to female ratio in the study group was 4:1 and the th

maximum incidence of acute myocardial infarction was seen in 6^{th} and 7^{th} decade.

2. In the study Hindus were 80% and Muslim were 20%.

3. In the study, the most common presentation symptom was chest pain

4. and is associated with sweat in 25% of patients and

5. breathlessness in 25% of patients and 6) palpitation in 10%.

7. In the study, the most common risk factor found was smoking followed

by hypertension and diabetes.

8. In the study group mean serum magnesium level in 60 patients on day-1 is 1.78±0.32 and on Day-5 is 2.32±0.44.

9. In the study group mean serum magnesium level in 30 patients with arrhythmia is 1.58±0.30 on day-1 and 1.96±0.32 on day-5.

10. In the study group, mean serum magnesium level in 30 patients

without arrhythmia is 2.10±0.50 on day-1 and 2.56±0.48 on day-5

SUMMARY

SUMMARY

Coronary artery disease is a major cause of morbidity and mortality throughout the world. Major cause of death in coronary artery disease are due to complications like arrhythmias.

In the present study, patients with acute myocardial infarction with low magnesium level are more prone to develop ventricular arrhythmias compared to those who are having normal magnesium levels. Magnesium replacement therapy in patients with acute myocardial infarction who is having low serum magnesium level may reduce the incidence of arrhythmias.

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. **PROFORMA**

Name:	Sl. No.	Unit
Age	IP No.	DOD
DOA	Sex	Occupation
Final diagnosis	religion	

Presenting Symptoms

1. Chest pain
2. Sweating
3. Dyspnoea
4. Orthopnea
5. PND
6. Palpitation
7. Others

History of present illness

1. Chest pain

* Location

* Nature	Severity	Mild/ moderate/ severe
	Character	Pricking/ throbbing
		Squeezing/ constricting
		Burning
		Agony/ other types

* Onset – Hrs/ days back

* Radiation

* Continuous/ intermittent

* Aggravating factor

* Relieving factor On taking rest

On taking drugs

2. Sweating

3. Dyspnea Grade-I II III IV

4. Orthopnea

5. PND

6. Palpitation

* Nature Continuous/ racing of heart beats

Other irregularities

* After excitement/ exercise/ drugs

* Periodicity

* Duration

7. Cough

* Dry/ productive

* Frothy sputum/ hemoptysis

8. Gastrointestinal symptoms

* Nausea

* Vomiting

* Dyspepsia/ heart burn/ indigestion

* Hiccoughs

9. Neurological symptoms:

* Convulsions

* Giddiness/ Fainting

- Time of onset after pain

- Duration

- Consciousness

10. Vascular symptoms

* Phlebitis

* Varicosity

* Claudication

* Raynaud's phenomenon

11. Endocrine symptoms

* Polyuria/ polydypsia/ polyphagia

* Excessive heat/ cold intolerance

* Any change in voice

* Change of hair distribution

12. Miscellaneous

* Swelling of the limbs

* Abdominal distension

* Visual disturbance

III Past History

1. Angina

    * Type

    * Duration

    * Frequency

    * Any treatment taken

2. Myocardial infarction

    * No. of attacks

    * Details of admission and treatment

3. Hypertension

    * Duration

    * Treatment – not taken/ regular/ irregular

    * Controlled/ Uncontrolled

4. Stroke/ TIA

    * Treatment

5. Diabetes mellitus

    * Duration

    * Treatment – Not treated/ regular/ irregular

    * Controlled/ Uncontrolled

6. Renal diseases

7. Peptic ulcer

8. Rheumatic fever

IV] Family History (Among-I, II, III Degree relatives)

1. IHD

2. Hypertension

3. Diabetes mellitus

4. Obesity

5. Cerebrovascular accidents

6. Sudden death

7. High cholesterol

V] Personal History

1. Socioeconomic status – Rich/ Middle/ Poor

2. Physical activity – Sedentary/ moderate exertion/ severe exertion

3. Marital status – Single/ married/ widow/er

4. Habits:

i) Diet Vegetarian/ non-vegetarian/mixed

ii)Alcohol Type

Quantity

Frequency

Since how long

iii) Coffee/ tea/ other beverages

No. of cups per day _____ since _____ years

iv)Smoking: Beedies/ Cigarettes/ Others

Quantity _____ _____ _____

Duration _____ _____ _____

v)Tobacco/ Pan chewing

5. Appetite

6. Bowels

7. Micturition

8. Sleep

9. Medication

   i) Corticosteroids

   ii) Diuretics

   iii)Others

10. Drinking water source

11. If female

i) Menstrual history

ii)Obstetric history

iii)Oral contraceptives

VI} General Physical Examination

1. Built: Well built/ dwarf/ average

   Height _____

2. Nutrition: Normal/ Obese/ undernourished

3. State of consciousness

4. Cyanosis

5. Clubbing

6. Anemia

7. Edema

8. Xanthoma/ Xanthelasmas

9. Arcus senilis/ Juvenilis

VII] Systemic Examination

1. Pulse

* Rate

* Rhythm

* Character

* Volume

* Condition of vessel wall – normal/ thickened

* Other peripheral pulses

2. BP

Upper limb

Lower limb

3. JVP

4. Epigastric pulsation

a) Inspection

-Shape of chest

-Apical impulse

-Other pulsations

b) Palpation

-Apical impulse

-Palpable heart sounds

-Parasernal heave

-Thrills

-Other pulsations

c) Percussion

-Left border of the heart

-Right border of the heart

-Upper border of liver dullness

-Any abnormal areas of dullness (Lt/ Rt I ics)

d) Auscultation:

Heart sounds: Normal/ accentuated/ muffled/ slit

* 1st sound

* 2nd sound

* 3rd sound

* 4th sound

Mitral area

Tricuspid area

Pulmonary area

Aortic area:

a) Extra sound: Irregular rhythm/ triple rhythm/ galloop rhythm

Murmur

Pericardial rub

b) Respiratory system

c) Gastrointestinal system

d) Genitourinary system

VIII] Investigation

1.Urine Albumin

Sugar

Micro

2. ECG at the time of admission

3. S.magnesium Sodium Potassium SGOT SGPT

1st day

5th day

IX] Course in Hospital

Clinical parameters ECG Management

X] Final remarks and the summary of the case:

KEY TO MASTER CHART

CVS.....Cardiovascular system

RS.....Respiratory system

mm of Hg.....Millimeter of mercury

BP.....Blood pressure

Mg2+ .....Magnesium

Na+ .....Sodium

K+ .....Potassium

mg/dL.....Milligram/ deciliter

meq/l .....Milliequivalent liter

VPCS.....Ventricular premature contractions

VT .....Ventricular tachycardia

ECG.....Electrocardiogram

CCF.....Congestive cardiac failure

MASTER CHART

No.	NAME	AGE	SEX	IP No	Symptom	Signs/ BP	ECG	DAYS	Mg	Na	k+	Complica
1	Jeyaraj	75	M	16184	Chest pain- 3 hr	s1s2+ BP=120/80	AWMI	15	1.51 1.90	136	3.8	VPCS recovered
2	Jeevanandam	31	M	17131	chest pain-6hrs	s1s2+ BP=110/70	ASMI	15	1.94 2.40	138	4.0	recovered
3	Kumeresan	58	M	17182	chest pain 4 hrs	S1S2+ BP=130/80	IWMI	15	1.87 2.12	136	3.8	VPCs recovered
4	Kendiyammal	80	F	17462	chestpain -6 hrs	S1S2+ BP=120/70	IWMI	15	2.12 2.60	140	4.0	VPCs recovered
5	Durairaj	50	M	17667	chestpain - 2 hrs	s1s2+ BP=80/50	AWMI	15	1.12	143	3.9	VT,died
6	Palpandi	37	M	17929	chestpain - 8 hrs	s1s2+ BP=130/70	IWMI	15	1.82 2.10	147	4.5	VPCs recovered
7	Kumeresan	38	M	18182	chestpain - 4 hrs	s1s2+ BP=110/70	IWMI	15	1.62 1.82	143	4.2	recovered
8	Kaliyamoorthi	45	M	18289	chestpain -10 hrs	s1s2+	ASMI	15	1.92 2.86	138	4.5	SVT recovered

No.	NAME	AGE	SEX	IP No	Symptom	Signs/ BP	ECG	DAYS	Mg	Na	k+	Complicat
9	Mani	65	M	18362	chestpain -3hrs	s1s2+ BP=110/70	IWMI	15	2.12 2.34	146	4.7	VPCs recovered
10	Raj	68	M	18409	chestpain -6 hrs	s1s2+ BP=140/80	AWMI	15	1.45 1.91	142	4.9	recovered
11	Ganesan	39	M	18824	chestpain -8 hrs	s1s2+ BP=??	AWMI	15	1.09	146	4.0	VT ,DIED
12	Ayyappan	70	M	19412	chestpain -4 hrs	s1s2+ BP=100/80	IWMI	15	1.45 1.68	138	4.8	recovered
13	Mathialagan	65	M	18942	chestpain -2 hrs	s1s2+ BP=100/80	ASMI	15	1.92 2.18	145	4.2	recovered
14	Rajkumar	58	M	20241	chestpain -6 hrs	s1s2+ BP=90/60	AWMI	15	1.39	140	4.8	VT died
15	Krishnamoorthi	62	M	20542	chestpain -8hrs	s1s2+ BP=130/80	IWMI	15	1.98 2.45	134	4.0	recovered
16	Valliyammal	82	F	20914	chestpain -4hrs	s1s2+ BP=120/80	ASMI	15	2.18 2.52	142	4.2	recovered

No.	NAME	AGE	SEX	IP No	Symptom	Signs/ BP	ECG	DAYS	Mg	Na	k+	Complicat
17	Muthurasi	65	F	21847	chestpain =8hrs	s1s2+ BP=160/70	AWMI	15	2.12 2.56	148	4.5	recovered
18	Basheer	52	M	21997	chestpain -10 hrs	s1s2+ BP=70/50	AWMI	15	1.18 1.65	142	4.8	Cardioge nic shock
19	Senthilkumar	48	M	22121	chestpain -2hrs	s1s2+ BP=150/70	IWMI	15	1.68 1.82	145	3.9	recovered
20	Tamilarasi	68	F	22456	chestpain -4 hrs	s1s2+ BP=90/70	IWMI	15	1.42 1.78	139	4.2	VT recovered
21	Kumaran	69	M	22679	chestpain -3hrs	s1s2+ BP=110/70	ASMI	15	1.83 2.18	141	4.5	VPCs recovered
22	Muhammed	53	M	22828	chestpain -4 hrs	s1s2+ BP=150/80	ASMI	15	1.95 2.12	148	5.0	recovered
23	Selvi	68	F	23167	chestpain -8 hrs	s1s2+ BP=130/80	AWMI	15	2.15 2.34	139	4.9	VPCs recoverd
24	Stalin	72	M	25826	chestpain -4 hrs	s1s2+ BP=140/90	AWMI	15	2.02 2.42	145	5.0	recovered

No.	NAME	AGE	SEX	IP No	Symptom	Signs/ BP	ECG	DAYS	Mg	Na	k+	Complicat
25	Muruganantham	48	M	26212	chestpain -8 hrs	s1s2+ BP=90/60	AWMI	15	1.42 1.84	145	3.8	LVF recovered
26	Kumari	65	F	26574	chestpain -6 hrs	s1s2+ BP=160/90	IWMI	15	1.56 2.28	146	4.2	VPCs recoverd
27	Veeraiyan	80	M	26589	chestpain -5 hrs	s1s2+ BP=180/90	ASMI	15	1.82 1.98	149	4.8	VPCs recovered
28	Jahangir	70	M	26987	chestpain -4hrs	s1s2+ BP=140/80	AWMI	15	2.09 2.43	143	3.9	recovered
29	Rajendhiran	60	M	27217	chestpain -2 hrs	s1s2+ BP=120/70	ASMI	15	1.22 1.58	134	4.0	VPCS Died
30	Amina	80	F	27861	chestpain -5hrs	s1s2+ BP=100/70	AWMI	15	1.76 1.98	136	4.2	recovered
31	Shahul	67	M	27921	chestpain -7 hrs	s1s2+ BP=160/90	ASMI	15	1.23 1.67	145	4.0	VPCs recoverd
32	Keerthana	78	F	27998	chestpain -4hrs	s1s2+ BP=??	AWMI	15	1.13	138	4.5	recovered

No.	NAME	AGE	SEX	IP No	Symptom	Signs/ BP	ECG	DAYS	Mg	Na	k+	Complicat
33	Rasu	80	M	28714	chestpain -6 hrs	s1s2+ BP=??	ASMI	15	1.23	135	3.9	VT died
34	Ramkumar	40	M	28843	chestpain -3 hrs	s1s2+ BP=160/90	IWMI	15	1.46 2.48	137	5.2	VPCs recoverd
35	Muhammed	60	M	29214	chestpain -5 hrs	s1s2+ BP=150/90	AWMI	15	1.52 2.34	141	3.8	SVT recovered
36	Abdulla	75	M	30672	chestpain -9hrs	s1s2+ BP=140/80	AWMI	15	2.19 2.65	133	3.6	recovered
37	Jayakumar	65	M	30754	chestpain -7 hrs	s1s2+ BP=90/60	IWMI	15	1.18	145	4.7	VPCS Died
38	Thangaraj	70	M	30761	chestpain -1hrs	s1s2+ BP=130/80	AWMI	15	1.66 1.95	143	5.0	VPCs recovered
39	Suresh	48	M	30858	chestpain -4 hrs	s1s2+ BP=150/90	AWMI	15	1.34 1.54	138	4.4	recoverd
40	Ravi	58	M	30992	chestpain -6hrs	s1s2+ BP=??	AWMI	15	1.45	138	4.5	VT died

No.	NAME	AGE	SEX	IP No	Symptom	Signs/ BP	ECG	DAYS	Mg	Na	k+	Complicat
41	Kiruthiga	70	F	31714	chestpain -4 hrs	s1s2+ BP=120/80	IWMI	15	1.93 2.74	139	4.9	recovered
42	Ramu	40	M	32543	chestpain -1 hrs	s1s2+ BP=100/70	IWMI	15	1.76 1.98	142	3.9	VT recoverd
43	Noorjahan	50	F	32594	chestpain -4 hrs	s1s2+ BP=110/60	AWMI	15	1.72 2.68	149	3.7	recovered
44	Alamina	65	F	32672	chestpain -2hrs	s1s2+ BP=80/60	IWMI	15	1.27	136	4.9	VPCs Died
45	Jayaraman	35	M	32765	chestpain -6 hrs	s1s2+ BP=100/60	AWMI	15	1.79 1.99	135	4.2	Recovered
46	Suhakar	56	M	33632	chestpain -9hrs	s1s2+ BP=110/80	ASMI	15	1.86 2.49	142	4.0	VT recovered
47	Revathi	50	F	33876	chestpain -3 hrs	s1s2+ BP=140/90	AWMI	15	1.34 1.54	140	4.5	recoverd
48	Sukumar	38	M	34543	chestpain -2hrs	s1s2+ BP=150/80	ASMI	15	1.75 2.23	137	3.9	VPCs recovered

No.	NAME	AGE	SEX	IP No	Symptom	Signs/ BP	ECG	DAYS	Mg	Na	k+	Complicat
49	Appusamy	68	M	35543	chestpain -7 hrs	s1s2+ BP=120/80	IWMI	15	1.43 1.84	138	4.8	Recovered
50	Rubina	50	F	35987	chestpain -1 hrs	s1s2+ BP=150/80	AWMI	15	1.76 2.18	135	4.9	Recovered
51	Riyaz	50	M	36345	chestpain -3 hrs	s1s2+ BP=110/70	AWMI	15	1.82 2.02	144	3.7	VPCs recovered
52	Jayakumar	65	M	37765	chestpain -6hrs	s1s2+ BP=90/60	AWMI	15	1.19 1.65	145	3.9	VPCs died
53	surya kumar	45	M	37675	chestpain -4 hrs	s1s2+ BP=100/60	AWMI	15	2.23 2.65	142	4.1	recovered
54	Thangamuthu	35	M	37776	chestpain -5hrs	s1s2+ BP=??	ASMI	15	1.10	140	5.3	VT DIED
55	Soundharya	58	F	38764	chestpain -2 hrs	s1s2+ BP=140/90	IWMI	15	1.74 1.96	139	4.6	recoverd
56	Krishnamoorthi	78	M	38742	chestpain -4hrs	s1s2+ BP=90/60	IWMI	15	1.94	135	3.9	VPCs died

No.	NAME	AGE	SEX	IP No	Symptom	Signs/ BP	ECG	DAYS	Mg	Na	k+	Complicat
57	Arumugham	70	M	39456	chestpain -3 hrs	s1s2+ BP=110/70	ASMI	15	1.93 2.64	138	4.1	recovered
58	Remya	60	F	40764	chestpain -6 hrs	s1s2+ BP=140/90	IWMI	15	1.76 2.08	142	5.1	VPCs recoverd
59	Annadurai	67	M	44685	chestpain -9 hrs	s1s2+ BP=80/60	AWMI	1	1.51	145	4.9	VT Died
60	Krishnan	54	M	45465	chestpain -2hrs	s1s2+ BP=80/50	AWMI	15	1.89 2.45	143	3.8	Cardiogenic shock recovered